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NERVOUS AND MENTAL DISEASES

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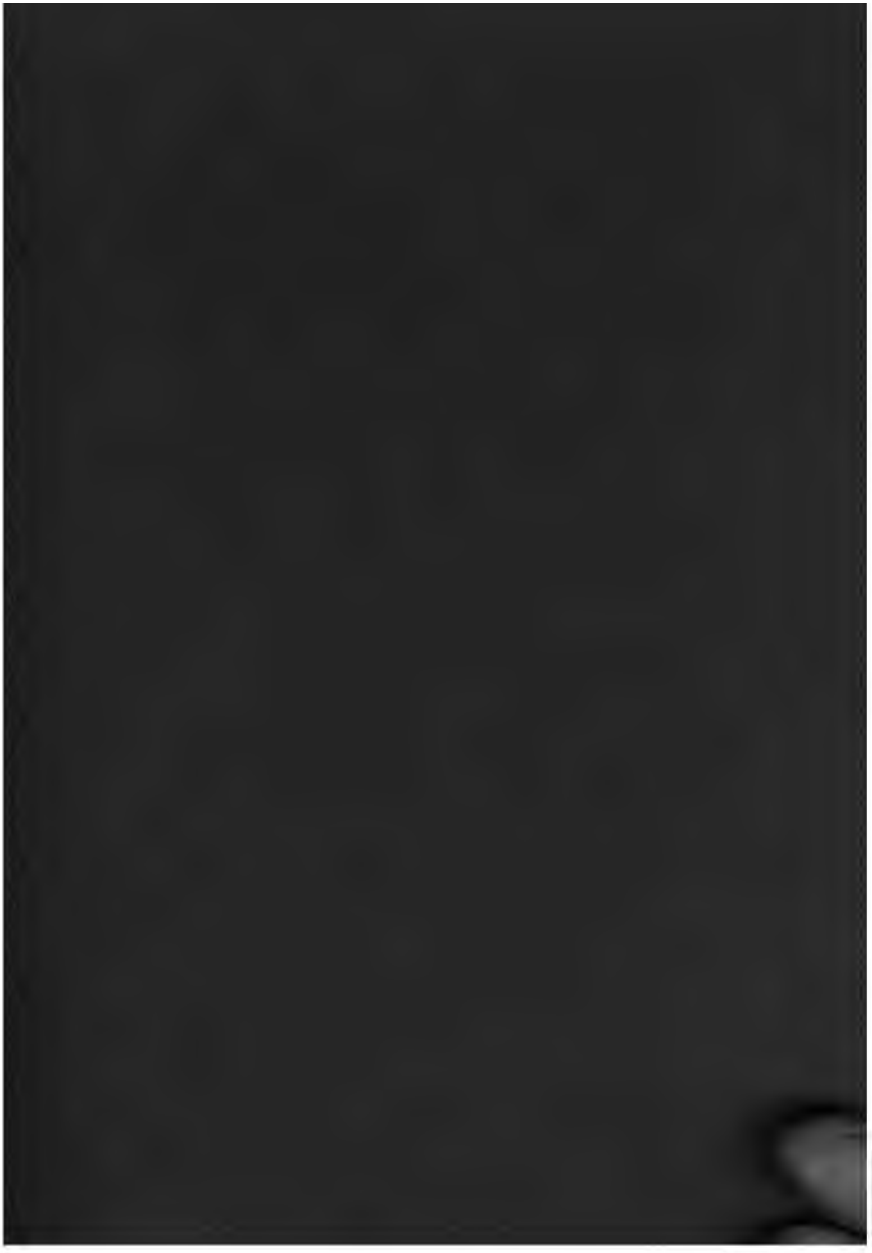

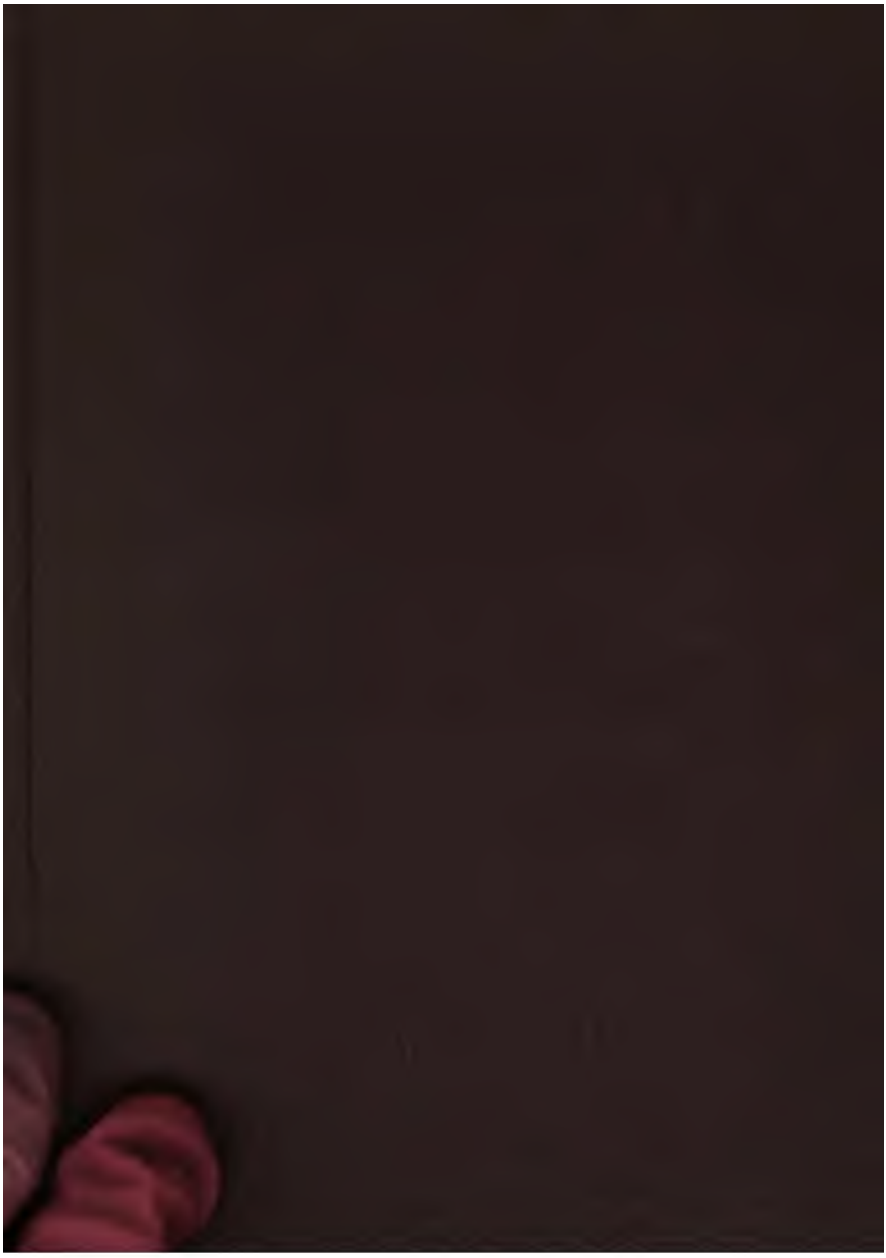

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The Medical Epitome Series.

NERVOUS AND MENTAL DISEASES.

A MANUAL FOR STUDENTS AND PRACTITIONERS.

With an Appendix on Insomnia.

BY

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AUTHOR'S PREFACE.

In this age of rapid progress and evolution of new theories and sciences the student of medicine, who in four years is supposed to master the intricate and varied details of his chosen profession, and the busy practitioner, who must still spend a good part of his time in research and study to keep abreast with the rapid strides of advance, both feel the daily need of a text-book which will give them the essence of the subject which they are pursuing. It is with this idea that the author has undertaken to gather the various facts and data contained in the numerous text-books and pamphlets on the diseases of the mind and nervous system, and to weave them into a compact fabric, easily studied by those who are in search of precise information.

There is scarcely a single author or lecturer of high standing whose teachings have not been incorporated in a condensed form into the pages of this volume. It is hoped that this work will accomplish what its author intends.

J. D. N.

New York, August, 1904.

EDITOR'S PREFACE.

In arranging for the editorship of *The Medical Epitome Series* the publishers established a few simple conditions, namely, that the Series as a whole should embrace the entire realm of medicine; that the individual volumes should authoritatively cover their respective subjects in all essentials; and that the maximum amount of information, in letter-press and engravings, should be given for a minimum price. It was the belief of publishers and editor alike that brief works of high character would render valuable service not only to students, but also to practitioners who might wish to refresh or supplement their knowledge to date.

To the authors the editor extends his heartiest thanks for their excellent work. They have fully justified his choice in inviting them to undertake a kind of literary task which is always difficult—namely, the combination of brevity, clearness, and comprehensiveness. They have shown a consistent interest in the work and an earnest endeavor to coöperate with the editor throughout the undertaking. Joint effort of this sort ought to yield useful books, brief manuals as contradistinguished from mere compends.

In order to render the volumes suitable for quizzing, and yet preserve the continuity of the text unbroken by the interpolation of questions throughout the subject-matter, which has heretofore been the design in books of this type, all questions have been placed at the end of each chapter. This new arrangement, it is hoped, will be convenient alike to students and practitioners.

V. C. P.

NEW YORK, 1904.

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NERVOUS AND MENTAL DISEASES.

PART I.

DISEASES OF THE PERIPHERAL NERVOUS SYSTEM.

CHAPTER I.

FUNCTIONAL DISORDERS.

Varieties.—The functional disorders of the peripheral nerves may be: (1) sensory, (2) motor, (3) of mixed form.

The **sensory disturbances** are paræsthetic neuroses (anæsthesia, hypæsthesia, and hyperæsthesia) and neuralgia.

Sensation may be diminished (hypæsthesia), or absent (anæsthesia), or increased (hyperæsthesia). In anæsthesia a strong irritation produces no reaction; in hyperæsthesia a weak irritation produces an exaggerated reaction; in hypæsthesia sensation is very imperfect.

ANÆSTHESIA OF THE SKIN.

Definition.—Anæsthesia of the skin is a partial or complete absence of sensation over a certain area of the cutaneous surface, due to interruption in the conducting power of the sensory nerves.

Cause.—Anæsthesia may be due to affection at the cutaneous end of the nerve (narcotics, injury, local disturbance of circulation), or to affection along the tract of the nerve (traumatism, inflammation, new growths), or to affection in the

spinal cord (locomotor ataxia, inflammation, new growths), or to affection of the cerebrum (hemorrhages in the internal capsule, hysteria, effect of anæsthetics on the brain). It may also be caused by infectious diseases, and sometimes by syphilis.

Symptoms.—Anæsthesia is rarely an independent affection, but mostly a symptom of other diseases of the nervous system. The patient usually notices that in some part of his skin sensation is not normal. He does not feel the pressure of his clothing, or small objects drop from his hand as he does not feel their presence. Anæsthesia may be combined with trophic disturbances. The patient is liable to receive injuries, scalds, burns, and cuts without being aware of the injury. In anæsthesia of the trigeminus nerve the face is usually bloated and the temperature on the affected side is somewhat lower than that of the other, the tongue loses its power of taste on the anterior two-thirds, and ulcers easily form on it from bites that are not felt.

The **prognosis** depends on the cause of the disease.

Treatment.—The treatment is directed to the removal of the primary cause. If the cause is obscure, a faradic or galvanic current may be used with good effect, or in cases of hysteria massage is very useful. The affected parts should be protected against injury. The use of strychnine internally is advised.

MOTOR NEUROSES.

Motor neuroses of the **various forms** are temporary or permanent paralysis and spasms and tremors occurring from overuse of the muscles (frequently associated with some form of "occupation" neurosis), the mixed neurosis of erythromelalgia (neuralgic pain and congestion in the feet) due to severe exertion or a sequel of a wasting disease, and Raynaud's disease (symmetrical affection of fingers or toes marked by angiospasm, coldness, pain, mottling, swelling, and finally gangrene).

NEURALGIA.

Definition.—Neuralgia is pain occurring in the course of nerves and in their area of distribution. The pain has remis-

sions and intermissions, and is due to some morbid affection of the nerves of sensation or their spinal or cerebral centres.

Etiology.—The affection may be idiopathic—depending upon some functional disturbance alone; or it may be symptomatic alone—due to some organic disease of the nerve or to some disease or pathologic state outside the nervous system. It occurs more frequently in women past the middle age, in those of a neurotic tendency, and in anæmic conditions.

Symptoms.—Pain is the chief and characteristic symptom. It may develop suddenly and without warning, or it may be preceded by soreness and stiffness in the tissues surrounding the nerve. In acute cases there is a burning or a violent tearing sensation in the course of the affected nerve increased on exertion. In others pain is intermittent or paroxysmal, of a darting, stabbing character, or accompanied by tingling sensations. There may be anæsthesia of the skin in the affected region or hyperæsthesia over the entire nerve-trunk with certain painful points (**points douloureux**). The attacks of pain may occur only at long intervals, but usually for some hours they occur every few minutes. In very severe cases pain may be continued for hours or days, and may persist in rare instances for months or years, being worse at a certain time each day (especially in malarial cases). Trophic and vasomotor disturbances are also met with, such as pallor or congestion of the affected part, various eruptions, changes in the color of the hair, and in advanced chronic cases symptoms of interference with the general nutrition. Severe paroxysms may be accompanied by spasms of adjacent muscles.

Diagnosis.—In severe cases it is difficult to distinguish between neuralgia and neuritis. In **neuritis** as a rule the pain is more constant in location, and it does not shift or dart from one nerve to another, and there are more muscular weakness and relaxation of tissues and the absence of history of repeated attacks.

Varieties.—Neuralgia may be classified, according to its **causation**, as neurotic, toxic, rheumatic, etc.; or according to its **location**, as trigeminal, intercostal, sciatic, and so on. Exposure to cold, mechanical irritation, tumors, aneurisms, press-

ure on nerves, and wounds may lead to neuralgia. It is more frequent in cold and damp climates than in dry and warm locations.

Exposure and reflex irritation from other diseases may precipitate attacks in those predisposed, or it develops without discoverable cause.

Pathology.—In most cases no pathologic changes in the nerve-fibres, cells, or ganglia may be detected. In some cases the nerve-trunks are swollen and tender, giving the pathologic appearance of mild interstitial neuritis.

Treatment.—Remove the cause if such is discoverable and can be reached. If dependent on anæmia, try to cure the constitutional complaint. Nutritious food, tonics, and hygienic treatment restore anæmic patients rapidly. In malarial, syphilitic, or gouty subjects use the constitutional treatment. In mild cases use counterirritants. The systematic use of galvanic electricity is the most valuable means at the physician's disposal, especially the descending current, beginning with the mild current and gradually increasing its strength. Internally arsenic, bromine, ergotine, aconite, gelsemium, valerian, ether, cannabis Indica, and quinine are recommended, although the latter often proves disappointing. In gouty subjects colchicine or ammonium muriate may be used. Alcohol often gives relief, but is a dangerous remedy. Nitroglycerin is useful in facial neuralgia. In severe forms the use of opium can not be dispensed with, but should be prescribed with caution. Acupuncture, injection of water beneath the skin, may also be used, although the actual cautery is far more efficacious. During the paroxysm coal-tar derivatives may be employed. Hydropathy (wet compresses, vapor baths, cold affusions, wet cloths, etc) is highly recommended. In cases in which no remedy seems to be of value neurectomy may be necessary. Sometimes neurotomy, or stretching of the nerve, is practised. In obstinate cases of facial neuralgia the Gasserian ganglion has been removed with success.

NEURALGIA OF SPECIAL NERVES.

Neuralgia of the Trigeminus.—Neuralgia of the fifth pair of cranial nerves is also known as **trifacial neuralgia, facial neuralgia, tic douloureux,** etc. This form is more frequent than all other forms of neuralgia combined, this nerve seeming peculiarly susceptible to functional and organic disorders. All three of the branches are seldom affected simultaneously, the ophthalmic branch being most often involved.

The **symptoms** depend upon the branch affected: (*a*) **Ophthalmic neuralgic pain** is of a supraorbital or frontal nature with a specially painful point at the supraorbital notch. Occasionally the pain is especially intense in the eyeball. (*b*) **Supramaxillary neuralgia,** with pain along the infraorbital nerve and a marked tender point at the infraorbital foramen. A toothache-like pain in the upper teeth is common. (*c*) **Inframaxillary neuralgia,** characterized by a diffused pain along the inferior dental branch, extending from the temporal region over the side of the face to the chin, with pain in the lower teeth and side of the tongue. The pain along the course of the trigeminus may come on without any special cause or after excitement of a physical or mental nature. In all forms trophic disorders may occur. The circulation becomes interfered with, and the face, at first pale, becomes red. Herpes may appear along the course of the nerve, while salivation and lachrymation are often prominent symptoms. Reflex facial muscular spasm (tic douloureux) may accompany the paroxysms, and is the most distressing form of nerve pain. The attacks may be very mild or very severe, and are sometimes sudden and epileptiform in character.

The affection should be **differentiated** from periostitis, osteitis, migraine, and toothache.

The **treatment** is directed toward the removal of the cause. Chronic cases are difficult to cure. Improve the general health. For the relief of acute pain opium is most reliable, but should not be employed until aconite or coal-tar remedies have been used. Galvanic electricity is very valuable. Quinine, arsenic, gelsemium, aconite, and strychnine or nitroglyc-

erin hypodermatically should be used. In obstinate cases operative interference is advisable.

INTERCOSTAL NEURALGIA.

Definition.—Neuralgia of one or more of the intercostal nerves.

Etiology.—Intercostal neuralgia may develop independently, in anæmia, after exposure to cold, from affection of the vertebræ, ribs, spinal cord, or from the pressure of tumors or aortic aneurism. It is more frequent in women, more common on the left side, and mostly in the nerves situated in from the fifth to the ninth intercostal space. If located in nerves distributed to the mammary glands, it gives rise to mastodynia, or neuralgia of the mammary gland. The fugitive pains of pleurodynia are to be regarded as neuralgic in character.

Symptoms.—The pain is usually very severe, especially on movement of the intercostal muscles. With the pain, as a rule, an herpetic eruption appears along the course of the affected nerve, which is supposed to be due to extension of the inflammation from the nerve-ends to the skin. Pain on pressure is most marked near the vertebral, the sternal end, and the middle of the nerve. The affection is very obstinate, and may continue long after the eruption has disappeared.

The **treatment** consists in the use of counterirritants, and in the more chronic cases electricity and anodynes are indicated. For herpes a protecting ointment is sufficient. In mastodynia applications of heat and electricity may be resorted to.

SCIATICA.

Definition.—A neuralgic affection of the sciatic nerve, characterized chiefly by pain along the course of this nerve.

Etiology and Pathology.—Sciatica may be regarded as a form of interstitial neuritis. The nerve-sheaths, and in severe cases the nerve-fibrils, are also affected. It is more common in middle life, and more frequent in men than in women. The gouty and those affected with muscular rheumatism are

more liable to the disease. The exciting causes are exposure to cold and wet, muscular overexertion, mechanical pressure from tumors, habitual constipation, and caries of the spine.

Symptoms.—Characteristic symptoms are pain and tenderness along the course of the sciatic nerve, with an accompanying weakness and a sensation of stiffness in the muscles. If sudden in its development, the patient experiences a lightning-like pain shooting downward from the sciatic notch, along the posterior surface of the thigh and the outside of the leg to the foot. These paroxysms are repeated on the slightest exertion, but after a while they become constant and dull, and worse at night. In most cases the onset of the pain is gradual, being at first felt only on exertion, but constant as it becomes more severe. There is pain on pressure above the hip-joint, at the sciatic notch, the middle of the thigh, behind the knee, below the head of the fibula, behind the external malleolus, and on the dorsum of the foot. Abnormal sensations (anæsthesia, hyperæsthesia, and paræsthesia) are noted along the course of the nerve. In the later stages the muscles supplied by the sciatic nerve become slightly atrophied, and may give abnormal electrical reaction. Trophic disorders, such as œdematous swelling and herpes, occur not infrequently. The affection may last for a few weeks or be prolonged for years, although eventually recovery is the rule. The more acute and severe the symptoms, the longer the case will probably last.

The affection **should be differentiated from** psoas abscess, lumbago, locomotor ataxia, and nervous coxalgia.

The **treatment** consists in absolute and complete rest of the limb, often necessitating immobilization of the leg by a long hip splint. In severe cases hot-water bags should be kept continually under the thigh from the sciatic notch to the popliteal space. Counterirritants, electricity (a strong descending current), and massage are useful in chronic cases. The hypodermatic use of drugs and injections of cold water into the muscles give great relief. Surgical measures may be tried, and the actual cautery may be used as a last resort. If dependent on some exciting causes, remedy those first. Recently local painting with guaiacol and glycerole, trinitrin, salophen

hypodermatically, compression, and deep injections of sodium glycerophosphate or antipyrin have been used.

OTHER FORMS OF NEURALGIA.

Lumbar neuralgia (involving the branches of the lumbar plexus).

Coccygodynia (neuralgia of the coccygeal plexus).

Neuralgias of the nerves of the legs (sometimes described as crural, plantar, metatarsal, and so on.

Cervicooccipital neuralgia (involving the first four pairs of spinal nerves).

Cervicobrachial neuralgia (involving the sensory nerves of the brachial plexus).

Occipital neuralgia (involving the occipital region).

Genital and rectal neuralgia (involving the genital or rectal nerves).

Their **etiology, symptoms, course,** and **treatment** are the same as of those of the other neuralgias as already described.

QUESTIONS.

What is neuralgia?
Give its etiology.
What are the symptoms of neuralgia?
Give differential points between neuralgia and neuritis.
What is the pathology of neuralgia?
What is the treatment of neuralgia?
Describe varieties of neuralgia met with.
What is neuralgia of the trigeminus?
What is intercostal neuralgia?
What is sciatica?
Define lumbago, coccygodynia, crural, plantar, metatarsal, cervicoöccipital, cervicobrachial, occipital, genital, and rectal neuralgia.

CHAPTER II.

VASOMOTOR AND TROPHIC DISTURBANCES OF THE PERIPHERAL NERVOUS SYSTEM.

Varieties.—The circulatory disorders most frequently met with are: (*a*) anæmia, (*b*) hyperæmia, (*c*) vasomotor paralysis, (*d*) vasomotor spasm.

ANÆMIA OF THE PERIPHERAL NERVES.

Anæmia **frequently accompanies** general anæmic states or is the result of obstruction of the bloodvessels of the nerves from atheroma or other causes. A positive **diagnosis** of this condition is very difficult and almost impossible as the **symptoms** are highly variable in character. Many of the neuritic pains and neuralgias and the pains accompanying the atheromatous changes of old age are due to anæmia.

Treatment.—The treatment must be directed to the removal of the primary cause with the use of general tonics and hygienic measures.

HYPERÆMIA OF THE PERIPHERAL NERVES.

Hyperæmia of the peripheral nerves does not give characteristic **symptoms** with the exception of muscular weakness, tenderness along the course of the nerve, and a darting neuralgic pain accompanied with sensory perversions not unlike the symptoms of a beginning neuritis. These symptoms may be **caused by** an extension of adjacent inflammation, exposure to cold, injuries, poisons, and all factors which tend to produce a simple neuritis.

The most effective **treatment** consists in cold applications, leeches, cupping, and counterirritation. In chronic cases tonics, potassium iodide, massage, and hydrotherapy may be employed.

VASOMOTOR PERIPHERAL PARALYSIS.

Vasomotor paralysis is **characterized by** an unnatural redness of the skin accompanied as a rule by a local elevation of temperature. The redness may be general or confined to certain parts of the body. This affection may accompany other neuroses or may occur independently as a result of an injury to the sympathetic nerves.

The **treatment** consists in the removal of the irritating **cause** if such may be done.

VASOMOTOR PERIPHERAL SPASM.

Vasomotor spasm is **characterized by** an unnatural pallor and coolness of the skin accompanied by stiffness and sometimes by pain. When of long duration, gangrene of the affected parts may develop. This is sometimes observed as a reflex result in those having their hands constantly in water (washerwomen).

These latter two conditions of supposed vasomotor disturbances are the result of interference with the functions of the vasoconstrictor and vasodilator nerves.

TROPHIC DISTURBANCES OF THE PERIPHERAL NERVES.

The existence of **trophic centres** is not proved. Some authors claim that typical disturbances are due to vasomotor irritation, but it is probable that there is a trophic centre.

Causes and Symptoms.—From an injury to the nerves often the skin becomes glossy or an abnormal amount of pigment is deposited. Atrophy of the muscles and the appearance of bedsores are also referable to trophic disturbances. The nails in some nervous affections become dark or crack, the hair is lost or turns white, and the trophic disturbances may even extend to the bones and joints. Associated with the typical disturbances are disturbances of the secretions and some neuroses. Sweating may be increased, diminished, absent, or unilateral. The saliva and other secretions may also be increased or diminished.

HEMICRANIA.

Synonym.—Migraine.

Definition.—Hemicrania is a peculiar form of severe paroxysm of unilateral headache associated with vasomotor disturbances and often accompanied by nausea and vomiting.

Etiology and Pathology.—There are no discoverable anatomic lesions. Some regard it as a neuralgic affection of athetotic origin of the fifth nerve; others as due to some vasomotor disturbances in the meninges; and others as a neuro-

sis, accompanied by periodic sensory discharges analogous to the motor discharges of epilepsy. It is more common in women than in men. It is often inherited, and may last from puberty to menopause, or during the whole lifetime. It is more common among the educated upper classes than among the laboring class. Mental emotion, physical or mental fatigue, constipation, a faulty digestion, lack of open-air exercise, disorders of the female genital organs, refractive errors, and ocular muscular insufficiencies, emotional outbursts, loud noises, toothache, rapid visual impressions act as predisposing or exciting causes. Some regard migraine as a toxic condition due to the absorption of toxins from the gastroïntestinal canal, and others regard it as a nervous diathesis due to anæmia and all conditions which weaken the resistance of the nervous system; others again as a neurosis due to insufficient excretion of the liver and of the kidneys.

Symptoms.—The premonitory symptoms, which may last a few hours or a day or more, are somnolent feelings of discomfort, uneasiness, malaise, chills, vertigo, visual disturbances, or disturbances of sense mechanisms. The real attack may follow quickly, beginning with the characteristic headache, at first unilateral, located in the temple, eye, or occiput, but spreading as it increases in intensity until it involves all of one side of the head and occasionally both sides. The pain is usually continuous and of great intensity, increased by movement, noises, light, or mental strain. The skin over the painful part is hyperæsthetic. There are loss of appetite, nausea, and vomiting. In the spasmodic form the affected side is painful, the skin is cool, the pupil is dilated, and the flow of saliva is increased. In the paralytic form the affected side is flushed, hot, the bloodvessels are dilated, and the pupils are contracted. There are great prostration and weakness and depression. Abnormalities may be present. The urine may be abundant or almost suppressed. The duration of the paroxysm may be from a few hours to several days.

The **prognosis** is not very good as the attacks are apt to recur in spite of treatment, which in many cases is without effect. In old age they usually cease, and in many women

there is complete cessation after the climacteric. The attacks in women are apt to occur at or near the menstrual periods. The general health does not seem to suffer as a rule, and life is not endangered or shortened by it. The prognosis is favorable in cases in which the source is removable, such as eye-strain.

Treatment.—A strict avoidance of all predisposing causes must be observed. In children of neurotic families special hygienic precautions must be observed, and all ocular disturbances rectified. During the attack absolute rest should be given. The most valuable drugs are coal-tar products, caffeine, sodium salicylate, guarana, ammonium chloride, bromides, chloral, cannabis Indica, antipyrin, antifebrin, bromide of gold, menthol, large doses of the three bromides, and methyl-blue. Amyl nitrite in the spasmodic form, ergotine in the paralytic form, are sometimes of benefit. Exercise great care in the use of chloroform and narcotics. The galvanic current, counterirritation, lavage of the stomach, large enemas, cold baths, massage, and the drinking of large quantities of water are very helpful adjuncts to other treatment.

NEUROMA.

Definition.—A morbid increase in the tissue-elements of the peripheral nerves.

Varieties.—False and true neuromata.

True neuromata are **composed of** medullated nerve-fibre or of nerve-tissue; and **false neuromata** are **composed of** other than nerve-tissue, being usually of secondary origin, extending to the nerve from adjacent structures (like fibroma, sarcoma, syphilis, and the tuberculous growths).

Symptoms.—The true neuromata may be hereditary or due to traumatism and amputation. True neuromata are often multiple and may give rise to no symptoms, or may cause a great deal of pain of an intermittent character which is increased on pressure when the conduction of the nerve-fibre is interfered with. Anæsthesia and loss of power may develop. Reflex spasm of a chronic or clonic nature may also occur in

the course of the disease. Sometimes it is possible to feel the little nodular growths, or when superficial they may be seen. These nodules may be very sensitive or give no pain at all. The disease is very apt to be chronic in duration, but may eventually disappear.

The **diagnosis** is often difficult except when the new growths are superficial.

Treatment in severe forms consists in extirpation of the new growths. If too numerous, common narcotics should be given for alleviation of pain.

False Neuroma.—Fibromata, sarcomata, myomata, syphilitic and leprous gummata are frequently called false neuromata, and any of these may give the same symptoms as the true neuromata.

NEURITIS.

Definition.—Neuritis is an inflammation of the nerves of an acute or chronic nature, associated with more or less degenerative change in the nerve-fibrils of the affected nerves.

Causes.—Neuritis may be produced by injury to the nerve, frequent muscular strains, exposure to cold, extension of inflammation from adjacent structures, pressure, compression in fractures, general infectious diseases, rheumatism, gout, syphilis, cancers, etc. In some cases it appears to be idiopathic.

Pathology.—In the **acute form** the vessels of the nerve become congested, exudation takes place, the nerve appears swollen and red. In severe cases the medullary sheaths and axis-cylinders of the nerve-fibres undergo destruction. These changes may take place along a considerable tract of the nerve, or be limited to certain spots. The process of destruction may stop, and regeneration, partial or complete, may take place. The regeneration consists in restoration of the axis-cylinder and in abundant formation of new connective tissue, and the growth of new fibres outward from the healthy stump into the old sheath.

When the morbid processes involve the nerve-sheaths and connective-tissue structures in particular, an "interstitial neuritis" results, and the changes are chiefly inflammatory in

nature. The connective tissue of the endoneurium and perineurium increases in volume; the nerve-fibres are compressed by the congested vessels and by the new tissue. When the disease locates itself in the nerve-fibrils, it gives rise to "parenchymatous neuritis," a condition resembling degeneration rather than true inflammation. There are congestion and exudation in the nerve-trunk, swelling, and degeneration of individual fibres.

In the **chronic form** the destruction is progressive from the beginning, advancing in many cases from the periphery toward the centre.

Varieties.—There are many varieties of neuritis based upon etiologic differences, severity of the disease, its distribution, etc., but the practically important varieties are neuritis following: traumatic neuritis resulting from direct mechanical injury of the nerve; neuritis following exposure to cold; neuritis due to extension of disease from adjacent parts (tuberculosis, cancer, syphilis, bone disease); neuritis resulting from bacterial poisons in the blood (typhoid fever, malaria, variola, diphtheria, etc.); neuritis resulting from action of poisons introduced from without (alcohol, lead, arsenic, mercury, etc.); neuritis of an endemic or epidemic form, as seen in tropical countries (beri-beri); and neuritis accompanying certain skin eruptions like herpes zoster.

If a single nerve of a small group of adjacent nerve-trunks is affected, the affection is called a "simple neuritis"; if a number of nerves be affected simultaneously, "multiple neuritis."

For the purpose of more simple classification the various forms are generally divided into "simple" and "multiple" neuritis.

SIMPLE NEURITIS.

Causes.—Simple neuritis may be caused by exposure to cold, involving in such cases nerve-trunks lying near the surface of the body, like "Bell's paralysis" or facial paralysis; by traumatism; by pressure due to morbid growths; aneurism;

sleeping with head resting upon the arms; the use of crutches, "crutch paralysis"; and by diseases involving the nerves by extension from adjacent affected parts.

Pathology.—The changes are localized to a limited portion of the nerve-trunk. At the point of injury the nerve-trunk is red, swollen, and infiltrated, occasionally surrounded by a gelatinous exudate. In mild cases the nerve-fibrils are very slightly, if at all, involved. In severer cases the nerve-fibrils give evidence of the alterations of a parenchymatous neuritis. In the severest cases the axial cylinders show degenerative changes. They become varicose, swell, disintegrate, and even entirely disappear, these changes necessarily involving all of the nerve-fibre lying below the seat of injury, although in rare cases they may extend upward. These changes may go on to complete destruction of the nerve-elements. If the original nerve injury be removed, regeneration begins very rapidly as a rule.

Symptoms.—Symptoms vary with the cause, nature, and location of the disease, although the main symptoms are the same in all cases, consisting in perversion, exaltation, and occasionally in entire abolition of the functions of the nerves involved. There is pain of a violent stabbing, darting nature along the course of the affected nerve, with tenderness to pressure along the trunk of the nerve. This pain may be very distressing, or may cause little inconvenience. Occasionally the skin over the affected nerve is swollen and red. As a result of impaired nervous function there is anæsthesia in the affected area, the muscles supplied by these nerves become weak and occasionally paralyzed, and in severe cases the muscles may become atrophied and a herpetic eruption often appears along the course of the nerve. The nutrition of the hair and nails is often affected, causing falling out or grayness of hair and loss of nails. The electrical reaction in milder cases is nearly normal; in severer forms there is a partial or complete reaction of degeneration. The duration depends upon the severity or curability of the initial lesion. The symptoms may disappear in a few days or may persist for months. Recovery is the rule if the primary exciting cause

can be remedied. Sometimes an acute attack is followed by the chronic form.

Treatment.—Remove at first the cause of the disease. After this, absolute and continued rest of the affected part must be insisted upon. Moist heat is a great relief for pain. Mild counterirritation by mustard or the galvanic current is advisable. In some cases ice is more effective than heat. Internally strychnine, salol, the salicylates, and the coal-tar derivatives may be used; in severe cases morphine should be given cautiously. Keep the bowels open by means of salines or mercurials. Improve the general health with tonics. Forbid all alcoholic stimulants. If tendency to chronic condition develops, use massage, faradic electricity, or change of climate.

MULTIPLE NEURITIS.

Synonyms.—Polyneuritis; Disseminated neuritis; Peripheral neuritis.

Definition.—Multiple neuritis is an inflammatory or degenerative disease of the peripheral nervous system, varying in extent and intensity, and affecting symmetric parts of the body.

Varieties.—The varieties arise from differences in nature, causation, severity, and location of the morbid process.

Causes.—1. Bacterial infection (diphtheria, typhoid, scarlet fever, measles, malaria, leprosy, beri-beri, la grippe).

2. Toxic substances in the blood (alcohol, lead, arsenic, carbon disulphide, copper, zinc, mercury, phosphorus, coal gas.

3. Anæmia and dyscrasic states (tuberculosis, syphilis, septicæmia, general malnutrition, diabetes).

4. Exposure to cold (rheumatic cases, overexertion).

Pathology.—The pathologic process is the same as in acute simple neuritis, and may be parenchymatous alone, or interstitial alone, or both.

Symptoms.—The affection usually begins suddenly with fever, loss of appetite, headache, and pains. In subacute cases there is no fever at the onset. The characteristic and

ever-present features are the abnormalities of nerve-reaction—*i. e.*, alteration of sensory, motor, reflex, and trophic functions of the nerves involved. In typical cases the onset is that of an acute infectious malady: chill, headache, pain in the back and limbs, loss of appetite, furred mouth, constipation, and other evidences of gastro-intestinal disturbance. The sensory symptoms are first to occur. Pain is usually sharp, severe, located in the limbs, increased by motion or pressure. There is general muscle- and nerve-tenderness; perversions of sensation now appear in the form of tingling, burning numbness, band about legs and body, diminution in tactile sense or hyperæsthesia, in rare cases anæsthesia. Ataxia manifests itself in delicate motions and in loss of position-sense. There are weakness, flabby condition of the muscles, and sometimes complete paralysis. Muscular weakness begins most frequently in the legs, extends upward, reaching the arms, and in typical cases the "wrist-drop" and "foot-drop" are soon apparent. Contractures may take place. Paralysis may reach the muscles of phonation, deglutition, respiration, resulting in impairment of these functions. Trophic disorders are frequent, like œdema, vasomotor paralysis, glossy skin, abnormal growth of nails and hair, profuse sweating, urticaria. The tendon-reflexes are usually diminished. There is no paralysis of sphincters. The electric current reacts the same as in simple neuritis, loss of faradic excitability, with anodal closure contraction greater than the reaction to kathodal closure.

The **intensity, rapidity of onset, course,** and **duration** vary considerably. In mild cases the symptoms disappear very soon. In severer cases the muscular symptoms persist for months. Deaths are not infrequent, due to failure of respiration or of heart action, or complicating diseases. In alcoholic neuritis a rapid onset is the rule with delirium and delusions. With a progressive development are symptoms of locomotor ataxia except bladder and rectal symptoms. The symptoms increase rapidly for three or four weeks, then after a stationary period of two months gradual recovery takes place at from six to twelve months

In **lead neuritis** symptoms may begin with intestinal colic, lead-line on gums, "dropped wrist." The recovery is gradual and poison is eliminated in about four months.

In **arsenical neuritis** the first symptoms are those of gastric disturbance, the legs and arms being equally affected. Recovery takes place in from two to six months.

In **diphtheritic neuritis** there is a severe paralysis of all extremities with sensory disturbance. Ataxia is marked in some cases. Recovery takes place in about three months.

Epidemic neuritis (beri-beri) is rarely seen save in the tropics.

Malarial neuritis usually gives symptoms of the idiopathic forms and occurs chiefly in malarial districts.

Syphilitic neuritis is chronic in its course, greatly resembling cases of tabes.

Tuberculous, rheumatic, septicæmic, and **diabetic neuritis** are also mentioned. Their nature is sufficiently indicated by their name.

Differential Diagnosis.—From **anterior poliomyelitis**: by pain, tenderness along nerves, sensory symptoms, and the symmetric distribution of paralysis. From **myelitis**: by absence of bladder and rectal symptoms, by absence of bed-sores and peripheral symptoms. From **locomotor ataxia**: by rapid onset and paralysis, the absence of bladder symptoms, and the absence of Argyll-Robertson pupil.

Prognosis.—Good in most cases, although recovery is slow.

Treatment.—First remove the cause, if possible, and relieve the pain and acute symptoms; after that use measures hastening regeneration of nerve- and muscle-fibres. Pain is controlled by hot applications, coal-tar derivatives, and opium. After acute symptoms disappear use massage and faradic electricity. Systematic exercise should be advised as soon as the muscles permit it. Strychnine, arsenic, iron, and quinine are general tonics.

In tachycardia cold applications to the chest give best relief. In paralytic cases passive movements are required. If contractures threaten, fixation must be resorted to; warm baths; attention to diet; absolute abstention from alcohol essential.

QUESTIONS.

What is anæsthesia of the skin?
Give its causation, symptoms, and treatment.
What are motor neuroses?
What varieties of vasomotor disturbances of the peripheral nerves are met with?
What is the treatment of such disturbances?
Define trophic disturbances of the peripheral nerves.
What is hemicrania?
What are the etiology and pathology of hemicrania?
What are the symptoms of hemicrania?
What are the prognosis and treatment of hemicrania?
What is neuroma?
Describe the varieties and symptoms of neuroma.
What is the treatment of neuroma?
What is neuritis?
What are the etiology and pathology of neuritis?
Give the varieties of neuritis.
Describe simple neuritis.
Give the pathologic changes and symptoms in simple neuritis.
What is the treatment of simple neuritis?
What is multiple neuritis?
What are the varieties of multiple neuritis?
Give the pathology and symptoms of multiple neuritis.
What are the differential diagnosis and the prognosis of multiple neuritis?
What is the treatment of multiple neuritis?

PART II.

DISEASES OF THE SPINAL CORD.

CHAPTER I.

DIAGNOSIS AND LOCALIZATION OF DISEASES OF THE SPINAL CORD.

THE RELATION OF THE CORD-SEGMENTS TO THE VARIOUS GROUPS OF MUSCLES, ACCORDING TO DR. M. ALLEN STARR (*published by courteous permission*).

Segment.	Muscles.	Reflex.	Sensation.
Second and third cervical.	Sternomastoid, trapezius.	Hypochondrium (?).	Back of head to vertex.
	Scaleni and neck.	Sudden inspiration produced by sudden pressure beneath the lower border of ribs.	Neck.
	Diaphragm.		
Fourth cervical.	Diaphragm.	Pupil, fourth to seventh cervical.	Neck.
	Deltoid.	Dilatation of the pupil produced by irritation of the neck.	Upper shoulder.
	Biceps, coracobrachialis.		Outer arm.
	Supinator longus.		
	Rhomboid.		
	Supraspinatus and infraspinatus.		
Fifth cervical.	Deltoid.	Scapular.	Back of shoulder and arm.
	Biceps, coracobrachialis.	Fifth cervical to first dorsal.	Outer side of arm and forearm.
	Brachialis anticus.	Irritation of skin over the scapula produces contraction of the scapular muscles.	Anterior upper two-thirds of arm.
	Supinator longus.		
	Supinator brevis.	Supinator longus.	
	Deep muscles of shoulder-blade.		
	Rhomboid, teres minor.	Tapping its tendon in wrist produces flexion of forearm.	
	Pectoralis (clavicular part).		
	Serratus magnus.		
Sixth cervical.	Brachialis anticus.	Triceps.	Outer side of arm and forearm
	Pectoralis (clavicular part).	Fifth to sixth cervical.	
	Serratus magnus.		
	Triceps.	Tapping elbow tendon produces extension of forearm.	Inside and front of forearm.
	Extensors of wrist and fingers.	Posterior wrist.	
	Pronators.	Sixth to eighth cervical.	
		Tapping tendon causes extension of hand.	

Segment.	Muscles.	Reflex.	Sensation.
Seventh cervical.	Triceps (long head). Extensors of wrist and fingers. Pronators of wrist.	Anterior wrist	Inner and back of arm and forearm. Radial distribution in the hand.
	Flexors of wrist. Subscapular. Pectoralis (costal part). Latissimus dorsi. Teres major.	Seventh to eighth cervical. Tapping anterior tendon causes flexion of wrist. Palmar, seventh cervical to first dorsal. Striking palm causes closure of fingers.	
Eighth cervical.	Flexors of wrist and fingers. Intrinsic muscles of hand.		Forearm and hand; median and ulnar areas.
First dorsal.	Extensors of thumb. Intrinsic hand muscles. Thenar and hypothenar eminences.		Ulnar distribution to hand.
Second to twelfth dorsal.	Muscles of back and abdomen. Erectores spinæ.	Epigastric, fourth to seventh dorsal. Tickling mammary region causes retraction of epigastrium. Abdominal, seventh to eleventh dorsal. Stroking side of abdomen causes retraction of belly.	Skin of chest and abdomen, in bands running around and downward corresponding to spinal nerves. Upper gluteal region.
First lumbar.	Iliopsoas. Sartorius.	Cremasteric, first to third lumbar. Stroking inner thigh causes retraction of scrotum.	Skin over groin and front of scrotum, and narrow band down the front of the thigh and leg.
Second lumbar.	Iliopsoas, sartorius. Flexors of knee (Remak). Quadriceps femoris.	Patellar tendon. Striking tendon causes extension of leg; second to fourth lumbar.	Inner side of thigh. Outer side of thigh.
Third lumbar.	Quadriceps femoris. Inner rotators of thigh. Abductors of thigh.		Inner side of thigh and leg to ankle.
Fourth lumbar.	Abductors of thigh. Adductors of thigh. Flexors of knee (Ferrier). Tibialis anticus.	Gluteal. Fourth and fifth lumbar. Stroking buttock causes dimpling in fold of buttock.	Inner side of foot.
Fifth lumbar.	Outward rotators of thigh. Flexors of knee (Ferrier). Flexors of ankle, peronei. Extensors of toes.	Achilles tendon. Overextension causes rapid flexion of ankle, called ankle-clonus.	Lower gluteal region back of thigh. Leg and foot, outer part.
First and second sacral.	Flexors of ankle. Long flexor of toes. Intrinsic muscles of foot.	Plantar. Tickling sole of foot causes flexion of toes and retraction of leg.	Leg and foot except inner side.
Third to fifth sacral.	Peroneus longus.	Bladder and rectal centre.	Perineum and back of scrotum; anus.

CHAPTER II.

DISEASES OF THE SPINAL MEMBRANES.

INFLAMMATIONS OF THE SPINAL MEMBRANES.

Varieties.—Inflammations of the spinal membranes may be divided into: (1) pachymeningitis, which may be either external or internal; and (2) leptomeningitis, which may be either acute or chronic.

EXTERNAL SPINAL PACHYMENINGITIS.

Definition.—A secondary inflammation of the outer layer of the spinal dura mater.

Etiology.—Caries of the spinal vertebræ, as in Pott's disease, and tumors or abscesses of neighboring structures pressing on the spinal column and causing an erosion of the vertebræ, are the main causes of this affection.

Pathology.—The involved area of the spinal dura is thickened and there is an increase of the connective tissues, and where the disease is secondary to tuberculosis of the vertebræ the dura may suppurate or become greatly thickened showing deposits of pus in place.

Symptoms.—These will vary according to the seat and extent of the inflammation. There are pain in the back radiating along the course of the spinal nerves involved, and tenderness over the diseased area of the spine. The muscles supplied by the nerves coming from the affected portion are rigid on account of hyperæsthesia, and if the function of the nerves is destroyed by pressure of the exudate or destructive inflammation, there are anæsthesia and paralysis. The spinal cord may be compressed in some cases causing spastic paraplegia and other pressure-symptoms.

Diagnosis.—Inflammation of the spinal membranes must be differentiated from myelitis: in the latter the onset is more decided and the paralysis is earlier; there is absence of the girdle sensation in pachymeningitis.

Prognosis is unfavorable except in cases that may be relieved by surgical interference, and in those caused by Pott's disease.

Treatment will depend upon the cause. If Pott's disease is the factor, it should be treated accordingly. The spine should be immobilized, followed by the occasional use of counter-irritation. The best hygienic surroundings are necessary; plenty of fresh air and sunshine. Tonics should be given.

INTERNAL SPINAL PACHYMENINGITIS.

Definition.—Internal spinal pachymeningitis is a chronic inflammation of the inner surface of the spinal dura mater.

Etiology.—The predisposing causes are male sex, middle life, prolonged exposure to cold, lowered vitality, traumatism, chronic alcoholism, syphilis, general paresis of the insane, and possibly rheumatism and gout.

Pathology.—The dura mater becomes very thick from a new growth of connective tissue, and the cord and nerve-roots become compressed, causing degeneration of nerves and muscles. The circumscribed form of the affection is apt to be in the cervical region.

Symptoms.—Hyperæsthesia and pain over the spine or at the periphery of the spinal nerves arising from the affected portion may be the first symptoms noticed. As the disease advances there is a slow development of paresis with atrophy and anæsthesia of the muscles supplied by these nerves. If the exudate compresses the cord, there is spastic paraplegia. If the compression involves the cervical region, all the muscles below that point are spastic and paretic.

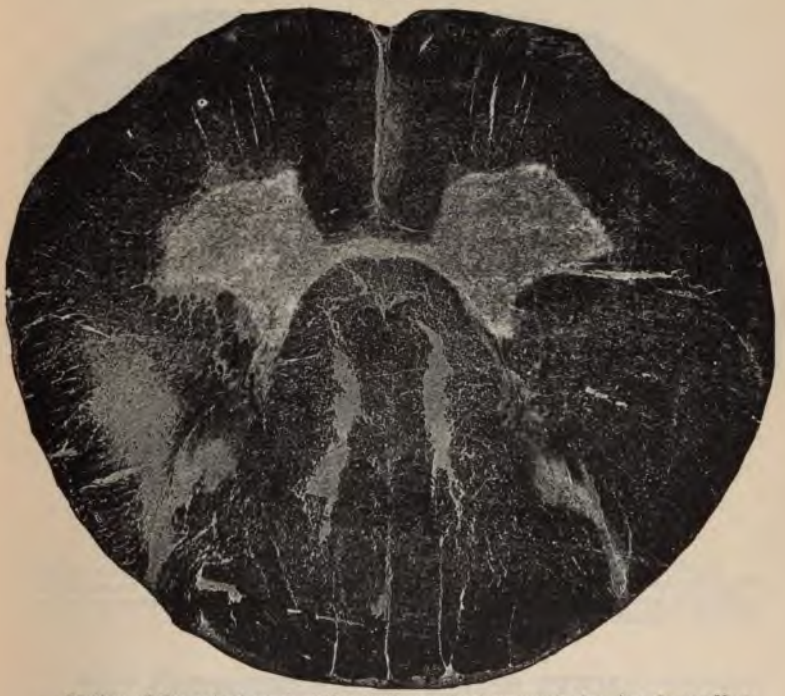
There is an increase of reflexes in the affected limbs. Bed-sores may develop. Duration of the disease is many years.

Diagnosis.—Internal spinal pachymeningitis must be differentiated from **myelitis**, which may be quite difficult. The pain of pachymeningitis is made worse by even slight movements, while that of myelitis is not influenced by movements of the body. The absence of incontinence of the bladder and rectum and the presence of more frequent rigidity and con-

tractures in pachymeningitis, will aid in diagnosing the affection from myelitis.

Prognosis.—This depends on the extent and cause. When the involvement is slight or is due to syphilis, the prognosis

FIG. 1.



Section of the spinal cord at the sixth cervical segment, showing descending degeneration in the left lateral pyramidal and the right anterior median columns after a small lesion in the motor tract of the right cerebral hemisphere. (Starr.)

should be guardedly favorable. In most cases the prognosis is bad.

Treatment.—Absolute rest. Tonics should be given. Counterirritation to the spine by strong tincture of iodine, blisters, or actual cautery. For the relief of the pain morphine,

phenacetin, or antipyrin may be required. In syphilitic cases, if the patient's condition will bear it, potassium iodide in conjunction with some mercurial should be given. In cases showing signs of a localized lesion surgical interference may be considered.

FIG. 2.

Section of the spinal cord at the fifth cervical segment, showing descending degeneration in the left anterior median and right lateral pyramidal tracts after extensive lesion in the left cerebral hemisphere. There is slight degeneration in the left pyramidal tract and in the posterior columns. (Starr.)

ACUTE SPINAL LEPTOMENINGITIS.

Definition.—Acute spinal leptomeningitis is an acute inflammation of the pia mater of the spinal cord.

Etiology.—Exposure to cold and wet, traumatism, the infectious diseases (pneumonia, typhoid fever, scarlet fever, yellow fever, septicæmia, pyæmia), direct extension of the inflammation from the cerebral membranes, syphilis, tuberculosis, and rheumatism are the etiologic factors.

Pathology.—The inflammation affects chiefly the pia mater, but the inner surface of the dura mater and the spinal cord itself may be involved. The membranes are opaque, congested, thickened, and adherent. There is an increase of

FIG. 3.

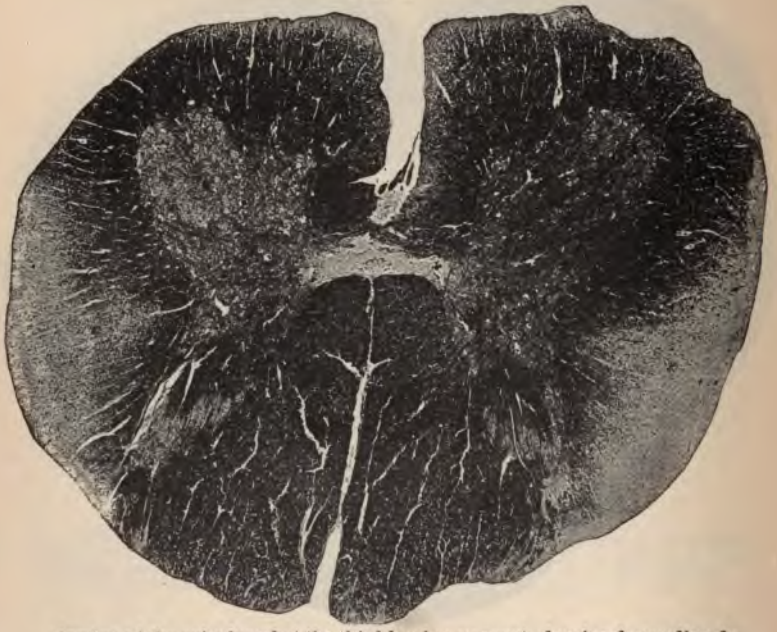
Section of the spinal cord (somewhat distorted) in the lower cervical region, showing bilateral descending degeneration in both anterior median and lateral pyramidal tracts. This section demonstrates the unequal size of corresponding columns on the two sides of the spinal cord. When the anterior median column is large in extent, the opposite pyramidal tract is correspondingly small. Integrity of the direct cerebellar columns is evident. (Starr.)

fluid in the arachnoid space, which may be either seroplastic or purulent lymph. The inflammation may finally attack the nerve-roots and the general surface of the spinal cord. There are a swelling and degeneration of the axis-cylinders of the

nerves, and in the cord there are proliferation of neuroglia-cells, infiltration with leucocytes, and granular degeneration of nerve-fibres, and the bloodvessels and their sheaths are dilated with leucocytes.

Symptoms.—The **prodromata** may be general malaise with

FIG. 4.



Section of the spinal cord at the third lumbar segment, showing descending degeneration in both lateral pyramidal tracts. A few fibres are degenerated in the anterior median columns. At this level there are no direct cerebellar columns. (Starr.)

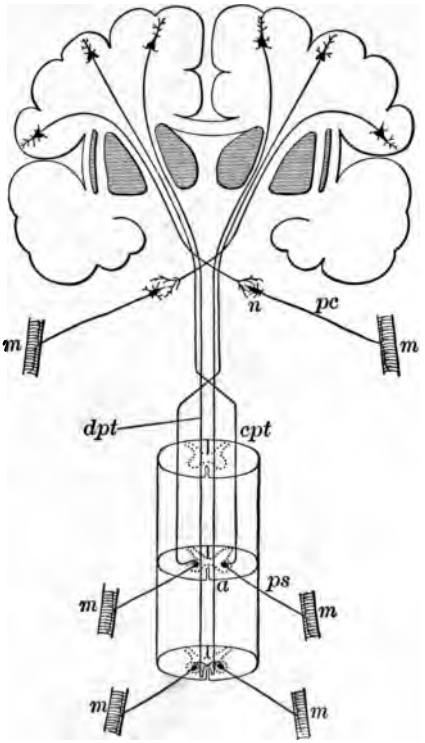
restlessness and sometimes vomiting for a few days prior to the onset.

The **onset** begins with a chill, followed by fever, vomiting, intense pain in the back radiating along the course of the nerves and tenderness along the spine.

Establishment.—These symptoms are soon followed by spas-

modic rigidity of the spinal muscles, sometimes sufficient to induce opisthotonos. The limbs are rigid and flexed upon the trunk. The respirations may be embarrassed by the spasm

FIG. 5.

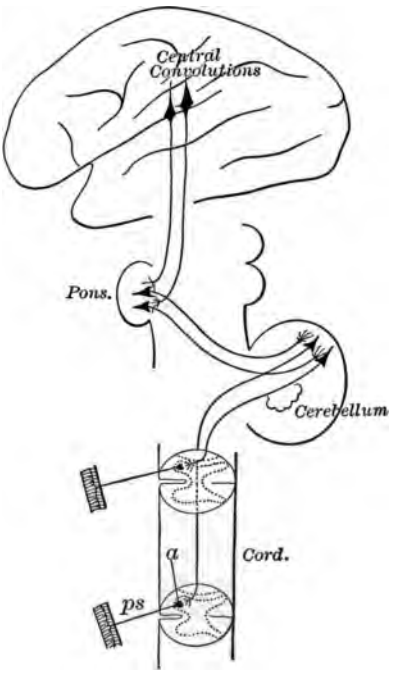


The direct or voluntary motor tract, showing the centre of the motor impulses from the cerebral cortex to the voluntary muscles: *m*, muscles; *n*, cells of nuclei of motor cranial nerves in pons and medulla; *a*, motor cells in anterior horns of the spinal cord; *dpt*, direct pyramidal tract; *cpt*, crossed pyramidal tract; *pc*, peripheral cranial nerve; *ps*, peripheral spinal nerve. (Van Gehuchten.)

of the chest muscles. The slightest movement of the body causes the patient great suffering. The temperature-range is from subnormal to 103° F.; the pulse may be rapid and

irregular, especially if the medulla oblongata is involved, when the respirations may be rapid, irregular, or of the Cheyne-Stokes type. The reflexes and sensation are increased at first. Paralysis may develop, whose type depends upon the location of the inflammation, which usually is widespread and involves

FIG. 6.



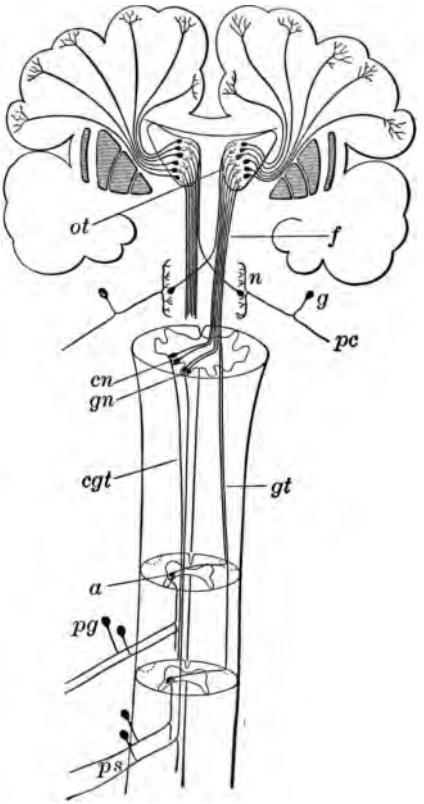
The indirect or involuntary motor tract.

nearly all parts of the spinal membrane. The most frequent type of paralysis is spastic paraplegia with paresis of rectum and bladder.

The course of the disease is from a few days to several weeks. If the patient lives, recovery is slow, and there is

generally some degree of paresis or paralysis which is often permanent.

FIG. 7.

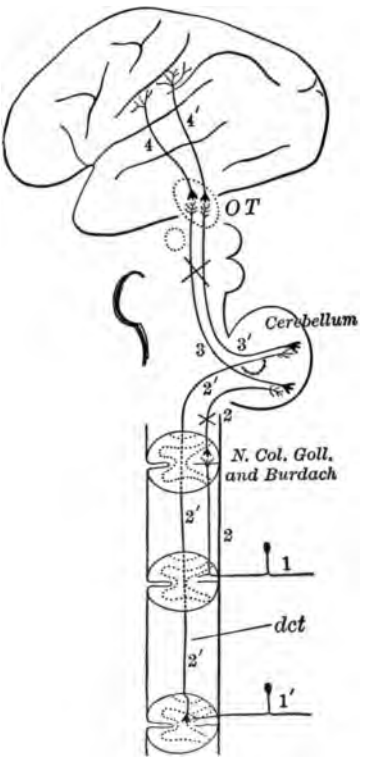


The direct sensory tract: *ps*, peripheral spinal nerves; *pg*, ganglion on posterior roots of the spinal nerves; *gt*, Gowers' tract; *cgt*, columns of Goll; and Burdach; *cn*, nucleus cuneatus: *gn*, nucleus gracilis; *a*, cells in posterior horn; *pc*, peripheral cranial nerve; *g*, ganglion on cranial sensory nerve; *n*, cells of cranial sensory nerves in medulla; *f*, fillet; *ot*, optic thalamus.

Diagnosis.—When the primary affection is severe, it may not be possible to diagnose the spinal affection during life.

Differentiation must be made from myelitis, subdural and intraspinal hemorrhage. In the beginning it is distinguished from **myelitis** by the paralysis and absence of marked pain in

FIG. 8.



Indirect sensory tracts: *dct.* direct cerebellar tract. The numbers represent the different series of neurons.

myelitis, and also by the variation in the pulse and temperature-range in acute leptomeningitis. It is diagnosed from **subdural** and **intraspinal hemorrhage** by the symptoms of the

latter coming on suddenly and always following some traumatic cause.

Prognosis is always serious, especially in the severer forms of the affection, and when the primary disease is dangerous to life and when the cervical region is affected. The prognosis in traumatic and syphilitic cases is more favorable and recovery may be fairly perfect.

Treatment.—Absolute rest in bed, the patient lying on the side or upon the abdomen with several pillows under him, exclusion of sound and light, and morphine hypodermatically to control the pain. Leeching, dry cupping, and counter-irritation by means of blisters or thermocautery along the spine should be used. Continuous application of the spinal ice-bag, if the patient can bear it. Frequent small doses of calomel, with bromides and chloral, should be given to diminish the spinal irritability. The hot bath or hot pack is frequently of great service in allaying pain. When syphilis or rheumatism is thought to be the cause, the antispecific or antirheumatic remedies should be given in full doses. Ergot and tincture of belladonna are recommended in the acute stage to contract the bloodvessels. In the subacute stage potassium iodide is of service to promote absorption. Mercurial inunctions and injections may be employed along the spine, which are of value in cases other than those due to syphilis, as the mercury acts as a counterirritant. Massage and electricity should also be used if the patient survive the acute stage.

CHRONIC SPINAL LEPTOMENINGITIS.

Definition.—A chronic inflammation of the pia mater of the spinal cord which is frequently a sequence of acute spinal meningitis.

Etiology.—Previous attacks of acute leptomeningitis, traumatism, syphilis, tuberculosis, chronic alcoholism, and (as a complication) myelitis are the etiologic factors.

Pathology.—The membranes are very thick and adherent, and fused into a homogeneous fibrous mass. The spinal cord may be sclerosed in places.

Symptoms.—Pain in the back, with shooting pain in the body and limbs, but not so severe as that in the acute form, with paræsthesia of the skin over the corresponding areas. The stiffness in the muscles is not so marked as in the acute form of the disease. The pain persists for weeks or months, the muscles atrophy, and the reflexes and sensation are completely lost. If the cord is compressed, the parts supplied by the affected nerves are paralyzed. The disease may exist for many years prior to death.

The **diagnosis** is often quite obscure. The affection must be differentiated from neuritis, progressive muscular atrophy, locomotor ataxia, and spinal caries.

Prognosis is best in cases following syphilis, but recovery following other causes is doubtful, the disease slowly progressing until death.

Treatment.—Absolute rest, a comfortable posture, and counterirritants to the spine are essential. Give sedatives for the pain. Potassium iodide should be given internally where the disease is suspected to be of syphilitic origin, and is of value in cases not due to specific origin. Tonics, tepid or cold baths, electricity, and massage may be used in the later stages.

MENINGEAL APOPLEXY.

Definition.—Meningeal apoplexy is a hemorrhage of large or small extent into and between the membranes of the spinal cord.

Etiology and Pathology.—Meningeal apoplexy may occur at all ages. Trauma, great physical exertion, severe convulsions, diseases of the vertebræ, meningitis, infectious and septic fevers, and aneurism are the etiologic factors. Extradural hemorrhage is slight in extent, and collects on the posterior surface. It occurs mostly in the cervical region. Intrameningeal hemorrhage sometimes fills the whole space between the cord and the dura mater, causing compression of the cord.

Symptoms begin suddenly, but cause no loss of consciousness. When the hemorrhage is of slight extent, the symptoms

are not marked; but when the hemorrhage is more or less extensive, symptoms of irritation in the sensory and motor branches of the corresponding parts occur as follows: severe pain, neuralgia in the extremities, muscular tremor and contractures. If the hemorrhage is extensive, symptoms of muscular paralysis and disturbances of the bladder functions may appear. An affection called **pachymeningitis interna hæmorrhagica** is sometimes met with, consisting of an encapsulated collection of blood on the inner surface of the dura mater, and occurring in the chronic insane and alcoholics.

The **prognosis** is favorable when the blood is rapidly absorbed.

Diagnosis may rarely be made with certainty.

Treatment.—There should be absolute rest, local application of ice to the spine, and bloodletting.

In chronic cases electricity, baths, and potassium iodide, to promote absorption, may be used.

QUESTIONS.

What is external spinal pachymeningitis?
What is the etiology of external spinal pachymeningitis?
What is the pathology of external spinal pachymeningitis?
What are the symptoms of external spinal pachymeningitis?
What is the diagnosis of external spinal pachymeningitis?
What is the prognosis of external spinal pachymeningitis?
What is the treatment of external spinal pachymeningitis?
What is internal spinal pachymeningitis?
What is the etiology of internal spinal pachymeningitis?
What is the pathology of internal spinal pachymeningitis?
What are the symptoms of internal spinal pachymeningitis?
What is the diagnosis of internal spinal pachymeningitis?
What is the prognosis of internal spinal pachymeningitis?
What is the treatment of internal spinal pachymeningitis?
What is acute spinal leptomeningitis?
What is the etiology of acute spinal leptomeningitis?
What is the pathology of acute spinal leptomeningitis?
What are the symptoms of acute spinal leptomeningitis?
What is the diagnosis of acute spinal leptomeningitis?
What is the prognosis of acute spinal leptomeningitis?
What is the treatment of acute spinal leptomeningitis?
What is chronic spinal leptomeningitis?
What is the etiology of chronic spinal leptomeningitis?
What is the pathology of chronic spinal leptomeningitis?
What are the symptoms of chronic spinal leptomeningitis?
What is the diagnosis of chronic spinal leptomeningitis?

What is the prognosis of chronic spinal leptomeningitis?
What is the treatment of chronic spinal leptomeningitis?
What is meningeal apoplexy?
What is the etiology of meningeal apoplexy?
What is the pathology of meningeal apoplexy?
What are the symptoms of meningeal apoplexy?
What is the diagnosis of meningeal apoplexy?
What is the prognosis of meningeal apoplexy?
What is the treatment of meningeal apoplexy?

CHAPTER III.

DISTURBANCES OF CIRCULATION IN THE CORD-TISSUE.

ANÆMIA.

Definition.—Anæmia of the spinal cord is a temporary or permanent diminution in the blood-supply.

Symptoms.—The condition is usually caused by a general narrowing of the arteries, as in chronic meningitis. If the diminution in blood-supply is permanent, paralysis necessarily follows. If the anæmia is transient (from arterial spasm), tetanoid symptoms often develop, as also the so-called "intermitting lameness." If the anæmia of the cord is part of a general anæmia, the spinal symptoms are not pronounced. There are dull pain and fatigue on slight exertion, and weakness which may increase to paralysis. Paraplegia may also result from anæmia, caused by excessive loss of blood.

Treatment.—Attention to the general health is most important in the treatment of these cases.

HYPERÆMIA OF THE SPINAL CORD.

Definition.—Hyperæmia is a temporary or permanent increase in the blood-supply.

Etiology.—Mechanical congestion may result from lying on the back. Active congestion may complicate many diseases, and may follow tetanus, strychnine-poisoning, general disturbances of circulation, and excessive coitus.

Symptoms are obscure and cannot be diagnosed during life, except when so active as to cause inflammation.

SPINAL APOPLEXY.

Definition.—A hemorrhage into the substance of the spinal cord of a primary or secondary nature.

Etiology and Pathology.—Primary hemorrhage is rare except after traumatism. Secondary hemorrhage may complicate myelitis, chronic alcoholism, sexual excess, tumors of the cord, and epidemic meningitis. The extravasation may be slight or severe. When large, the substance of the cord is destroyed in the direction of the long axis.

Symptoms.—Most cases begin suddenly, though occasionally there are prodromata, as disturbances of sensation. When the hemorrhage is extensive, there is a rapid development of paralysis, most marked in the lower extremities. There are also great pain in the back, paralysis of the bladder, anæsthesia, and changes of reflexes—symptoms the occurrence of which will depend on the location of the hemorrhage. If the blood is absorbed, the paralytic symptoms gradually disappear, but often the symptoms persist and death ensues.

Diagnosis.—Apoplexy of the spinal cord must be differentiated from multiple neuritis, hemorrhagic myelitis, and meningeal hemorrhage.

Prognosis.—The danger to life is always grave, but the prognosis is better when the dorsal region is the seat of hemorrhage.

The **treatment** consists of absolute rest, ice to the spine, laxatives, and ergot. The paralysis is treated according to general principles.

FUNCTIONAL DISTURBANCES OF THE SPINAL CORD.

Definition.—A set of symptoms resembling severe spinal disease, but having no known anatomic basis.

Etiology.—It is not known whether disturbances of the sensory tract or of the circulation are the basis of these

troubles. Severe emotional excitement, mental exertion, excessive use of tobacco or alcohol, and onanism are among the exciting causes. Hypochondriasis also may lead to functional disturbances.

Symptoms.—They are slow in their onset, beginning with fatigue and weakness, pain in the back of a more or less severe character, numbness in the extremities, sexual disturbances, and a number of general symptoms due to the neurasthenic condition of the patient. There may be tenderness along the vertebral column. The reflexes and sensations are usually normal, but there may be coldness, sweating, and chilly feelings. The appetite is good as a rule.

Diagnosis and Prognosis.—It is sometimes difficult to diagnose the affection from serious spinal disease except by thorough physical examination. Permanent recovery may ensue, but in some cases the affection, though never dangerous, continues through life.

Treatment.—Moral treatment is of chief importance. Proper diet, suitable exercise, electricity, cold baths, and tonics are of great benefit. Removal of any exciting factor is of pre-eminent importance.

QUESTIONS.

What disturbances of circulation in the cord are met with?
What are the symptoms of anæmia?
What are the symptoms of hyperæmia?
What is spinal apoplexy?
What is the etiology of spinal apoplexy?
What is the pathology of spinal apoplexy?
What are the symptoms of spinal apoplexy?
What is the diagnosis of spinal apoplexy?
What is the prognosis of spinal apoplexy?
What is the treatment of spinal apoplexy?
What are the functional disturbances of the spinal cord?
What is the etiology of functional disturbances of the spinal cord?
What are the symptoms of functional disturbances of the spinal cord?
What is the diagnosis of functional disturbances of the spinal cord?
What is the prognosis of functional disturbances of the spinal cord?
What is the treatment of functional disturbances of the spinal cord?

CHAPTER IV.

INJURIES OF THE SPINAL CORD.

CONCUSSION OF THE SPINE.

Definition.—A severe jarring of the body followed by a group of spinal symptoms supposed to be due to some minute changes in the cord of an unknown nature. It is probable that the condition is one of laceration.

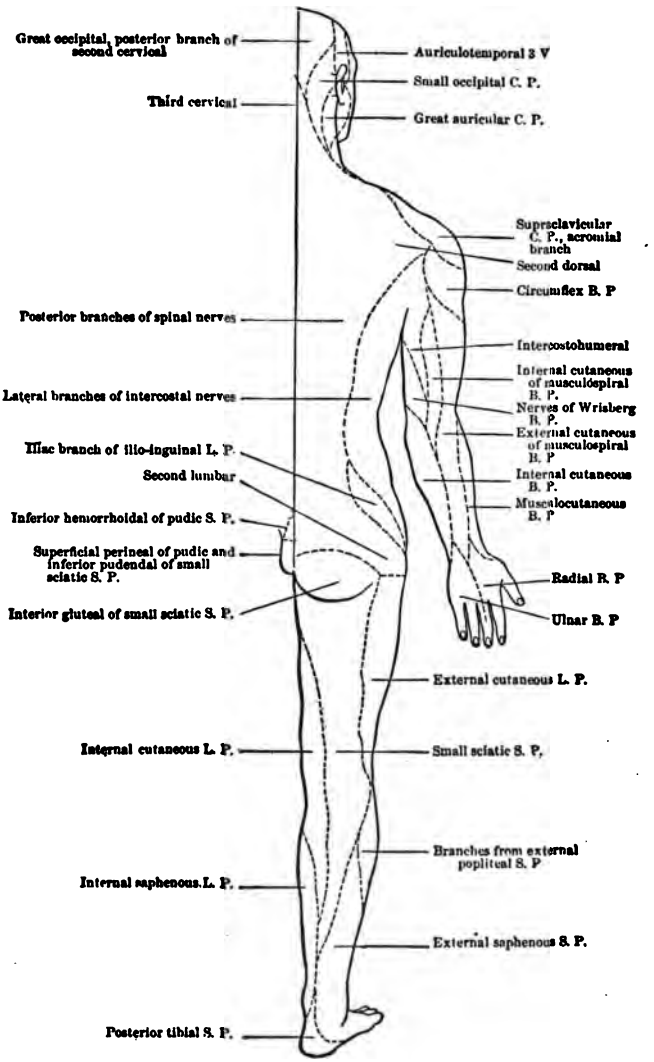
Etiology.—Severe concussions may result from railway accidents—"railway spine"—or violent bending of the body, fall from a horse, blow on the back, high jumping, etc.

Symptoms.—In some cases the onset of the symptoms is sudden, due to a jarring of the brain as well as of the spinal cord, loss of consciousness, complete paralysis, small pulse, collapse, and within a few hours death. In others the severe symptoms are followed by gradual improvement, but it is often many years before recovery is complete. There are a difficulty in locomotion and weakness in the upper extremities, but the electric reaction is normal. There are pain of varying degree and tenderness on pressure along the spine. Sometimes sensation is diminished as well as the reflexes. Cerebral symptoms, such as headache, dizziness, fainting, etc., may be present or absent. Anomalies in the action of the bladder, rectum, and sexual organs may or may not occur. These symptoms may be absent for months or years. In other cases there are no special symptoms after the concussion, but within a few weeks or months there is a gradual development of spinal symptoms, combined with bulbar symptoms and cerebral disturbances. These cases gradually get worse, although recovery may take place.

Diagnosis.—Concussion must be differentiated from neurasthenia or hysteria.

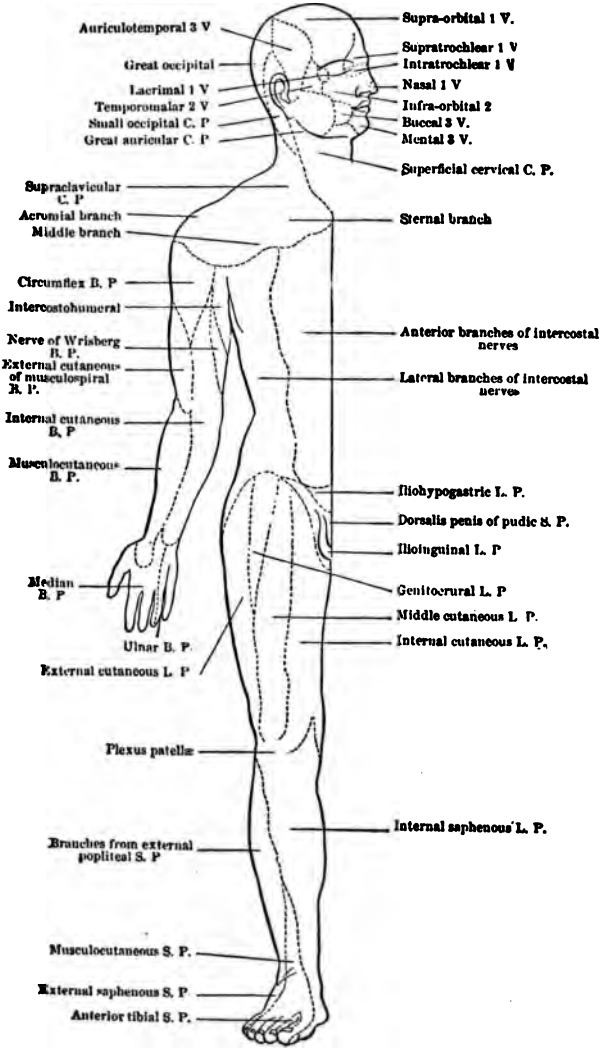
Treatment.—The treatment should comprise rest, stimulants, electricity to counteract the shock to the system. In chronic cases electricity, potassium iodide, ergot, strychnine, baths, and change of climate are recommended.

FIG. 9.



Cutaneous distribution of nerves. (After Flower.)

FIG. 10.



Cutaneous distribution of nerves. (After Flower.)

TRAUMATISM OF THE SPINAL CORD.

Etiology.—There may be compression or laceration of the cord in fractures and dislocations of the spine. The cord may be injured by gunshot- and stab-wounds. In these there may be laceration of the vessels, producing hemorrhage, which may compress the cord sufficiently to cause paraplegia or hemorrhage into the substance of the cord itself, or there may be laceration of the cord. In gunshot-wounds the cord more often suffers from displaced fragments of bone than from the ball itself.

Pathology.—The extent of injury varies greatly according to the cause. There is usually hemorrhage, either outside or on the inner surface of the dura mater, or the pia mater, or into the cord itself, and with more or less laceration of the cord-tissue. As a result of the effusion the cord usually softens; the nerve-fibres waste and degenerate in an ascending or descending manner.

The **symptoms** differ according to the seat of injury. There are usually marked motor and sensory disturbances, and occasionally sudden complete paralysis of the upper or lower extremities. The bladder and rectum may be abnormal in their functions. There are pains and abnormal sensations. The temperature is often increased, the reflexes are diminished. When the damage to the spinal cord is extensive, death ensues sooner or later. The symptoms in some cases are very slight in the beginning, but increase in severity within a few days, or there may be a sudden increase in them a few months subsequently. In other cases the symptoms, at first marked, gradually abate, which is due to absorption of the blood-clot.

Prognosis.—If the symptoms are slight, a recovery may be expected; but immediately after an accident a guarded prognosis should be given even if the symptoms are slight, as grave signs may develop in the course of a few days. The sooner symptoms occur as a rule, the better is the prospect of ultimate improvement or recovery, provided they are not so severe as to be incompatible with life.

Treatment.—In the beginning the treatment of these cases,

and frequently many of the later cases in which the spinal column is injured, is purely surgical. In gunshot-wounds, if the compression is caused by fragments of bone, blood, or the bullet, the pressure should be relieved by operation. In injury due to any cause where there are symptoms of compression, it should be relieved by surgical interference. In all cases of spinal injury, however slight the symptoms, absolute rest is necessary, and should be required for some days or weeks according to the severity of the beginning symptoms. The symptoms of myelitis should be treated accordingly. If there is muscular wasting, the nutrition of the muscles should be maintained by electric stimulation. Whenever there is evidence of products of secondary inflammation outside of the cord, trephining should be considered. The neuralgic condition of spinal pain and tenderness which frequently follows injury should be treated by the use of counterirritation (as the actual cautery, blisters) or iodine; and the use of sedatives, as morphine, cannabis indica, etc., but the latter remedies should be used as seldom as possible.

QUESTIONS.

What is concussion of the spine?
What is the etiology of concussion of the spine?
What are the symptoms of concussion of the spine?
What is the diagnosis of concussion of the spine?
What is the treatment of concussion of the spine?
What is the pathology of traumatic lesion?
What are the symptoms of traumatism of the spinal cord?
What is the prognosis of traumatism of the spinal cord?
What is the treatment of traumatism of the spinal cord?

CHAPTER V.

ORGANIC DISEASES OF THE SPINAL CORD.

CAISSON DISEASE.

Synonym.—Divers' paralysis.

Etiology.—An affection occurring in divers, in bridge-builders, and others subjected to increased atmospheric press-

ure. The symptoms develop on coming to the surface suddenly, where the atmospheric pressure is greatly lessened.

Pathology.—The affection is supposed to be due to the presence of gases in the blood, having been absorbed during the exposure to the high pressure, and escaping thence into the nerve structures of the cord and causing an arrest of nervous functions from pressure. By others it has been ascribed to stasis of blood and œdema. The cord is congested and sometimes hemorrhages are found.

The **symptoms** usually occur on return to the surface of the water or after the lapse of several hours. There are pains in the ears and joints and bleeding from the nose. The pulse is slow and strong. Gastralgia and vomiting often occur. There are disturbances of motor and sensory functions. Paraplegia or hemiplegia may occur, usually beginning suddenly. Sensation may also be lost. Retention of urine, partial or complete, generally exists. Occasionally cerebral symptoms, like loss of consciousness, coma, and irregular breathing, develop, and death follows in a few hours. In most cases recovery takes place in a few days or a few weeks.

Treatment.—As a preventive persons engaged in work under water should be advised to change gradually from a great depth to the surface, and not go into the outer air suddenly. When the disease is once developed, it should be treated in the same way as an acute myelitis, which it resembles in its symptoms.

PRESSURE MYELITIS.

Definition.—An inflammation of the spinal cord, due to compression of the cord from the presence of new growths and diseases of the vertebræ.

Etiology and Pathology.—Accumulation of masses of inflammatory products within the membranes; chronic caries in tuberculosis and other disease; new growths; aneurism; and cancer of the vertebræ may by compression of the cord produce paralysis of the parts below the seat of affection. It occurs in children as well as in adults. In tuberculous caries

of the vertebræ, having its seat most commonly in the dorsal portion, several vertebræ become diseased, and, being rendered softer, the healthy vertebræ compress them, and, pushing them toward the cord, the spinal canal is narrowed. From encroachment of the vertebræ the cord is compressed. The cord at this point is considerably narrowed and softened, of a flat, cylindric, or indented appearance. The nerve-fibres are not destroyed, though their power of conduction is interfered with by the pressure. In some cases there is very little evidence of inflammation in the cord itself, though sometimes there is a disintegration of the axis-cylinder and the neuroglia and an increase in new connective tissue, while in other cases the symptoms of inflammation are very marked. In chronic cases the degeneration extends in an ascending or descending direction.

The **symptoms** depend on the degree of the compression, its duration, and the amount of degeneration produced. In some cases caries may be present for a long time without ever involving the spinal cord. In other cases there first are the signs of the presence of caries of the vertebræ, followed by slowly or quickly developing spinal symptoms. In these cases there is pain in the affected part of the spine, increased on exertion, and there is also pain shooting along the course of the compressed nerves. The pains are of a neuralgic character, and are constant or intermittent. There are hyperæsthesia and later anæsthesia. The muscles become weak and atrophy; the paresis increases, going on to paralysis if the affection is progressive. As to the reflexes, when the compression is above the reflex arc for the lower extremities, the tendon-reflexes are greatly increased, as the inhibitory influences do not reach the reflex arc; the cutaneous reflexes are not so markedly increased, and are often even diminished. There are sometimes trophic and circulatory disturbances. In severe cases the functions of the bladder and rectum are interfered with.

Prognosis and Diagnosis.—In tumors (cancer, etc.) the prognosis is always grave. In caries recovery may take place even in cases seemingly severe and hopeless, except when some

complicating disease arises. This affection should be **differentiated** from subacute transverse myelitis, new growths within the cord, and extensive pachymeningitis.

Treatment.—If this condition is due to spondylitis, that affection should be treated in the proper manner. Rest in bed, cupping, and counterirritation of the spinal column, especially the Paquelin cautery, electricity, the internal use of iodine, are all recommended. The other symptoms are treated as are those of ordinary myelitis. Orthopædic appliances are often followed by great benefit in suitable cases.

MYELITIS.

Definition.—Myelitis is an inflammation of the spinal cord.

Varieties.—Acute, subacute, and chronic myelitis according to the **onset** and **course.** The **anatomic division** includes the cervical, dorsal, and lumbar varieties; the transverse; the diffuse or disseminated; the focal; the central; the marginal; the parenchymatous and the interstitial myelitis.

Etiology.—The disease may occur at any age. It is more common in males than in females. The exciting causes are prolonged exposure to severe cold, excessive physical or mental exertion, sexual excess, trauma, hemorrhage into the cord, alcoholic excess, acute infectious diseases, syphilis, and purulent inflammations of the neighboring organs. Arsenic, lead, and other metallic poisons may induce the disease.

Pathology.—The membranes are usually infected and opaque. The bloodvessels are engorged and increased in number. Capillary hemorrhages are occasionally present. The cells are swollen and the nuclei displaced. Later there appears a marked increase of connective tissue with destruction of the nervous tissue and ganglion-cells. The bloodvessels become thickened and dilated; the nerve-tissue is almost completely displaced by connective tissue. There is a disappearance of the cells and they are replaced by granular and amorphous material. The pia and dura may be involved. The most frequent seat of the affection is in the dorsal portion of the cord. In some instances, especially those where syphilis is

the cause, there are a complete softening and breaking down of nerve-tissue in the cord.

Symptoms.—The symptoms depend on the seat and extent of the inflammation. The **onset** may be abrupt, subacute, or gradual. When the onset is abrupt, there may be a chill followed by fever (101°–103° F.), general malaise, loss of appetite, coated tongue, and constipation. The essential nervous symptoms are usually irritation at first, although motor and sensory paralysis may be present from the beginning. These nervous symptoms depend upon the locality and extent of the myelitic process, so a certain degree of familiarity with the topographic anatomy and functional localization of the cord is necessary. The most common irritative symptoms are hyperalgesia and hyperæsthesia. The patient may complain of pain in the back radiating into the limbs, with the various forms of paræsthesia as numbness, tingling, etc. There may be retention, or the urine may dribble involuntarily. These bladder symptoms may complicate myelitis in any part of the cord, but mostly in affections of the posterior columns. There is usually obstinate constipation; less frequently there is incontinence of feces. Sexual power is diminished or lost. There is frequently a sense of painful constriction—"girdle pains"—at the level of the disease, this being a guide to the level of the cord-lesion. If the disease is of the cervical region, involving the origin of the brachial plexus, the arms will be affected, and there may be an optic neuritis. Should the disease extend upward, involving the vagus, there is dyspnœa with circulatory and vasomotor symptoms. Paralysis follows the irritative symptoms, and may become more or less complete. There is anæsthesia, and all forms of common sensation may be impaired or lost. This paralysis may be flaccid or spastic with abolished or exaggerated reflexes according to the location of the disease. Widespread motor and sensory paralysis may follow slowly a prolonged irritative stage, or it may become extensive very rapidly after the onset of the disease. Later there is atrophy of the affected muscles, with or without degeneration. In the spastic cases there may be a great contracting of the affected muscles, the knees being

flexed upon the abdomen or the heels touching the buttocks. Bedsores develop in the later stages of the disease. Most cases are chronic in their course, lasting one or more years.

The irritative symptoms are not so pronounced in the chronic form. The mind is usually not affected.

In all the forms, when the trophic nerve-fibres become involved various nutritional changes occur in the region and organs supplied by the segments of the cord affected.

Diagnosis from **acute poliomyelitis**—in the latter there is absence of bladder and rectal symptoms, and there are no sensory disturbances.

From **Landry's disease**—in this there is absence of bladder and of rectal symptoms and of trophic disturbances; the loss of sensation is slight, there is no "girdle pain," and the disease is usually more rapid in its course.

From **meningitis** by the absence of the "girdle pain," of involvement of the sphincters, and the irritative phenomena are more marked than the paralytic.

From **locomotor ataxia**—the Argyll-Robertson pupil is absent in myelitis, as well as other ocular and optic nerve-changes, the pains are different in character and degree. There is an absence of true motor paralysis in locomotor ataxia.

Myelitis is diagnosed from **tumor of the cord** by the slower rate of progress in the latter, but the differential diagnosis is frequently quite difficult. It is diagnosed from **hemorrhage into the cord** by the paralysis developing rapidly in the latter.

Prognosis depends chiefly on the cause. If due to trauma, when the cause may be removed by surgical procedure, or due to syphilis, the prognosis should be guardedly favorable. Improvement and remissions occur, but recovery is rare.

Treatment.—The details of the treatment depend on the cause. Rest in bed, counterirritation, or wet cupping, but with great care on account of causing bedsores; a water-bed from the first may prevent this complication. In cases of retention of urine the catheter should be employed under the strictest possible asepsis. If pain is severe, it should be relieved by opiates. If syphilis is the cause, employ anti-

syphilitic treatment to the extent of tolerance, and in all cases of doubt mercurial inunctions should be resorted to. When due to trauma, abscess, or tumor, operative interference should be considered and decided promptly to prevent extensive secondary softening.

In tuberculous myelitis lumbar puncture with drainage at times gives great relief.

Sodium salicylate, small doses of mercury, or full doses of iron, may be given, in addition to the familiar local measures, during the acute stage.

In the chronic cases the galvanic may be alternated with the faradic current, and the use of massage and baths adopted. Internally, ergot, strychnine, silver nitrate, arsenic, and phosphorus have been used, but with little success. A tentative course of treatment with potassium iodide should be given in all chronic cases, because syphilis is so often the precursor of this form.

DISSEMINATED SCLEROSIS.

Definition.—A chronic affection of the spinal cord and brain, due to a dissemination of sclerotic patches in various parts of the central nervous system.

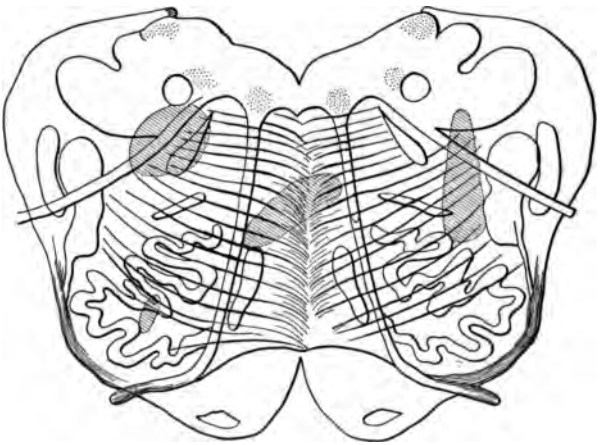
Etiology.—The causes which lead to other scleroses of the cord may induce the disease. Heredity, syphilis, mental or physical overexertion may have an influence on the development of the sclerotic nodules. The infectious fevers may be a cause.

Pathology.—There are small gray nodules distributed all over the cord and brain, chiefly in the white substance. Each patch consists of connective tissue, a number of fat-cells, with very few nerve-cells. There is hardly ever any secondary destruction in the cord itself.

The **symptoms** differ as the posterior or lateral columns are affected, depending on the location of the patches. The most constant symptom is a tremor resembling that of paralysis agitans, but in sclerosis tremor occurs only with intentional movement (volitional tremor), and is not regular in character, and is more marked in the upper extremities than in the

lower. There are nystagmus (tremor of the eyeballs); slow, hesitating speech; vague pains. Paresis does not occur until late in the disease. The tendon-reflexes are greatly increased, especially in the lower extremities. Sensory and cutaneous reflexes remain quite normal. The gait is usually dragging. The following cerebral symptoms are usually present: mental impairment, melancholia, dementia, apoplectic attacks, vertigo,

FIG. 11.



The shaded areas represent the sclerotic patches seen in the brain and cord in disseminated sclerosis of the central nervous system. (Starr.)

and epilepsy. Trophic disturbances and affections of the bladder and rectum are generally absent.

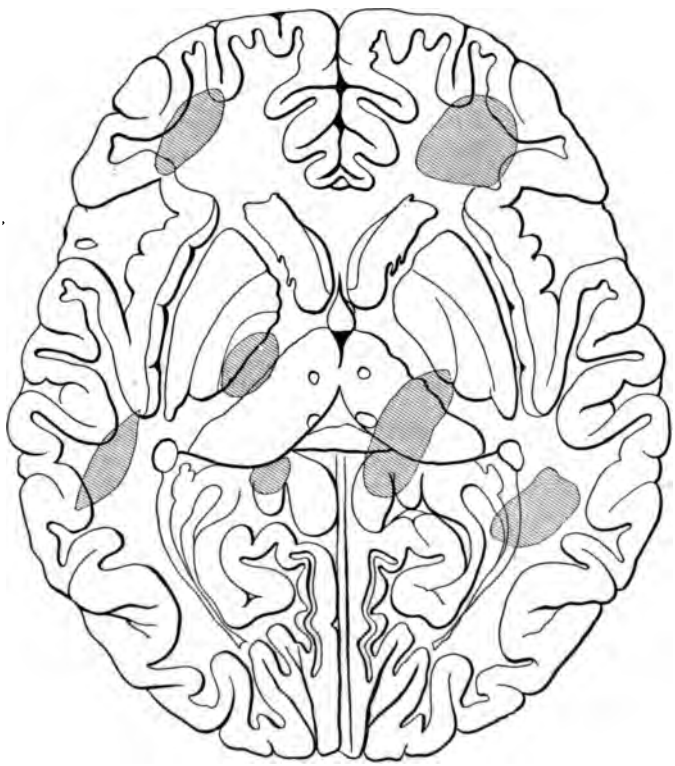
The disease is very chronic in its course, lasting for years and ending in death. The foregoing set of symptoms is not the exact type of disseminated sclerosis, as there are very many varieties resembling any of the affections of the spinal cord, because the sclerotic nodules may form at any possible point of the nervous system.

Diagnosis.—This disease may be mistaken for **paralysis agitans**, but the latter occurs later in life, the tremor is fine,

rarely involves the head, and is not made worse by use of the muscles; nystagmus is absent.

Prognosis is unfavorable. The duration is indefinite, and long remissions with improvement of the symptoms may occur.

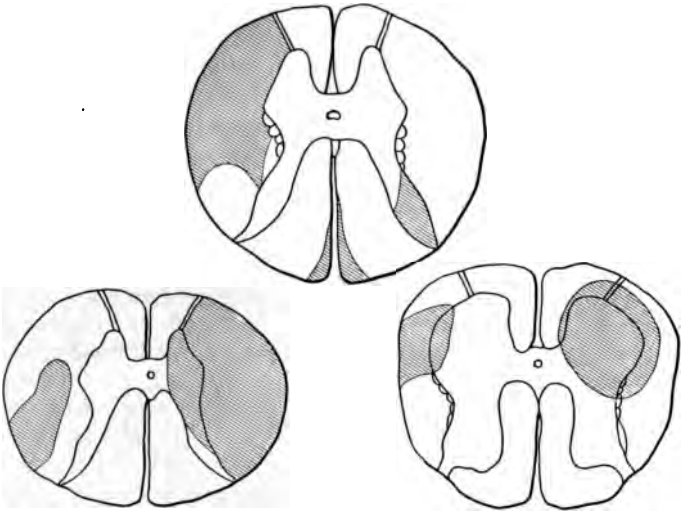
FIG. 12.



The shaded areas represent the sclerotic patches seen in the brain and cord in disseminated sclerosis of the central nervous system. (Starr.)

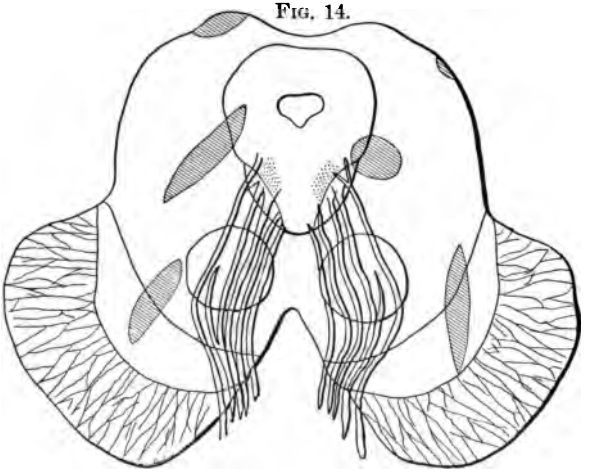
Treatment is that of chronic myelitis. Bromides, hyoscine bromate, hyoscyamine, and belladonna have been recommended for the tremors.

FIG. 13.



The shaded areas represent the sclerotic patches seen in the brain and cord in disseminated sclerosis of the central nervous system. (Starr.)

FIG. 14.



The shaded areas represent the sclerotic patches seen in the brain and cord in disseminated sclerosis of the central nervous system. (Starr.)

LOCOMOTOR ATAXIA.

Synonyms.—Tabes dorsalis; Posterior spinal sclerosis.

Definition.—A sclerosis affecting the posterior columns of the spinal cord, and characterized by incoördination, loss of deep reflexes, disturbances of nutrition and sensation, and various ocular phenomena.

Etiology.—It is a disease of adult life, persons under twenty-five years of age rarely being affected, and is more common in men than in women (10 : 1). It is sometimes met with in children the victims of hereditary syphilis. The chief predisposing cause is syphilis, which precedes it in from 70 to 85 per cent. of the cases according to various authorities. Exposure to cold and wet, alcoholic and sexual excesses, mineral poisoning, and great physical exertion are the exciting causes.

Pathology.—The membranes over the posterior columns are often opaque and adherent; the posterior columns have a gray, translucent appearance, and are atrophied. Microscopic examination reveals atrophy of the nerve-fibres and an overgrowth of connective tissue. The degeneration is most marked in the lumbar portion of the cord, affecting there the middle and posterior portions of the posterior columns; in the dorsal portions the whole of the posterior columns is usually affected; while in the cervical portion mostly the columns of Goll degenerate. The degeneration is usually symmetric in the two halves of the cord. Degenerative changes are frequently observed in the basal ganglia and in the peripheral nerves.

The **symptoms** of the disease are very numerous, but appear in succession and with the same regularity, making the diagnosis of this affection comparatively easy. They may be classified according to the time of their appearance: the prodromata, or stage of pain; the stage of ataxia; and the stage of paralysis.

The **prodromata** consist of lightning-like pains in the lower extremities, numbness, formication, sensation of dead extremities, "pins and needles" in the soles of the feet and fingers, coldness, itching of anus and scrotum or of other parts, a sensation of constriction around the chest, and headache.

Pain in the small of the back and loins of aching character may occur. These symptoms may constitute the only evidence of locomotor ataxia, and last for years; but sooner or later there are added absence of patellar reflex and an immobility of the pupil. The loss of knee-jerk, called Westphal's symptom, is always observed, and is due to a degeneration of the centripetal portion of the reflex arc in the middle portion of the posterior column of the spinal cord. The pupil fails to respond to light while it still accommodates for distance (Argyll-Robertson pupil). Contracted pupil "**myosis spinalis**," is frequent, but not constant. There may also be a paralysis of the ocular muscles, either on one or on both sides, coming on rather suddenly and dependent on degeneration of the respective nerves. In some cases there is also an optic atrophy which may begin quite early in the disease, ending in total blindness. There also may be a slight loss of cutaneous sensation. There may be imperfect control of bladder. Slow urination or slight dribbling and hasty urination. Later, control is very imperfect and may be painful. Cystitis may occur, and is a dangerous complication. There is usually obstinate constipation. There is loss of sexual desire, but this is occasionally preceded by priapism and sexual excitement. All these symptoms may last for several months or years.

The **ataxic stage** commences with disturbances of motion. The disturbances of coördination (ataxia) are very marked, especially in the lower extremities; the gait becomes difficult and uncertain; there is difficulty in rising or rapid turning; the legs are wide apart, feet lifted too high and come down too forcibly; the length of steps is irregular and the body imperfectly balanced. If the patient stands with feet together and eyes closed, he begins to sway (Romberg's symptom), which is due to a defect in controlling the muscles from impairment of sensation. There may be imperfect use of the hands in dressing, writing, etc. The definite cause of ataxia has not yet been ascertained, but it is probably due to a lesion of the gray matter. The power of the muscles is usually preserved in locomotor ataxia. The electric reaction of the muscles and nerves remains normal, but the muscles soon

become flabby from disuse. Sensation is changed from the beginning, and the lancinating pains are marked in all cases and come on in paroxysms. The pains are mostly in the legs, but also occur in the arms and head, the loins, the back, and the trunk. Herpes accompanies these neuralgic pains sometimes. Very soon anæsthesia develops. At first the tactile sense is interfered with; then the senses of pressure and temperature are diminished or lost. The muscular sense is greatly interfered with, especially when the controlling power of the eyes is temporarily taken away. Occasionally delayed sensations are observed, as is proved when the prick of a pin is not felt until a few seconds after being applied. This stage may last for many years, and may show a standstill; but it is usually progressive and advances to the third stage.

The **third** or **stage of paralysis** is marked by a gradual change to the worse, and the patient is unable to leave his bed. Paresis and even paralysis may occur from extension of degeneration to the lateral pyramidal tracts of the cord. Anæsthesia of the lower and sometimes of the upper extremities becomes marked. The joints (mostly the knee- and hip-joints) show on both sides of the body a painless swelling, from the presence of great quantities of serum. Spontaneous dislocations and fractures occur. Other trophic disturbances are rare, except bedsores and peculiar perforating ulcers of the sole of the foot. Control of the bladder is completely lost, and urine dribbles constantly. Death occurs from exhaustion, bedsores, cystitis, or complicating pneumonia.

There are often **complications** in other organs. There may be attacks of sharp pain, called "**crises**," coming on suddenly, in paroxysms. "**Gastric crises**" consist of violent pain in the stomach, vomiting, and vertigo, lasting a variable time. "**Intestinal crises**" consist of a very painful diarrhœa. "**Laryngeal crises**" are severe attacks of dyspnœa, due to a spasm of the glottis and associated with a severe paroxysmal cough. "**Cardiac crises**" are attacks of angina pectoris. "**Renal crises**" resemble attacks of renal colic. There are also **rectal**, **urethral**, and **testicular crises**, characterized by acute pain in the regions mentioned. Cerebral symptoms are sometimes

met with in the last stages of the disease, such as dementia and delusional insanity. Occasionally the sense of hearing is lost from degeneration of the auditory nerve.

Early diagnosis is reached by means of pains, fatigue, loss of knee-jerk, and Argyll-Robertson pupil. **Late diagnosis** is made by ataxia and bladder disturbances. In persistent "rheumatic pains," ocular disturbances, and gastric attacks the reflexes should always be examined. From **multiple neuritis**: the pain is not lancinating like that of ataxia, and the Argyll-Robertson pupil is absent. From **tumor of the cerebellum**: in this condition the reflexes are not abolished, lightning-like pains are absent, and instead there are persistent vomiting, headache, and optic neuritis.

Prognosis.—The affection is usually fatal, though it may last a great many years; arrest and even improvement are not infrequent.

Treatment.—Any cause that may hasten the production of the disease should be removed, such as mental or physical exhaustion, exposure to cold, alcoholic excess, and smoking. In most cases an antisyphilitic treatment should be begun at once—potassium iodide in full doses. In other cases potassium iodide in small doses, mercury, and arsenic are the most reliable remedies.

The patient should be placed under the best hygienic conditions; the diet nutritious but easily assimilable. Tonics are often indicated. Silver nitrate, strychnine, ergot, and phosphorus are recommended. The ascending electric current, massage, vapor baths, cold baths, mud- and iron-baths are valuable adjuvants to internal treatment. Counterirritation may be employed to the spine by means of the blister or actual cautery. Nerve-stretching and stretching of the spinal cord by means of an extension or suspension apparatus have been used with benefit in many cases. For the pain and the crises narcotics are used—antipyrin, phenacetin, etc. The laryngeal crises may be relieved by the inhalation of chloroform or amyl nitrite; the trophic disturbances by rest and apparatus; optic atrophy by strychnine.

In employing strychnine in this disease great caution should

be exercised as the dose is increased, because several fatalities have resulted from ordinarily therapeutic amounts.

HEREDITARY ATAXIA.

Synonym.—Friedreich's disease.

Definition.—Hereditary ataxia is a degenerative disease of the spinal cord, affecting the posterior and lateral columns, occurring in childhood and in several children of the same family beginning with symptoms resembling locomotor ataxia.

Etiology.—The greatest number of cases develop within the first decade of life. The affection occurs more frequently in boys than in girls, and has been noticed more in the rural districts than in the cities. It may frequently be traced to heredity, which is generally indirect, but is sometimes direct. Syphilis, epilepsy, alcoholism, and insanity are given among the causes of the ancestral taint. Several children in the same family may have the affection.

Pathology.—Sclerosis of the posterior and lateral columns of the cord.

Symptoms.—In very young children it is noted that the child is slow in learning to walk; it staggers in attempting to stand or walk; it uses its hands clumsily and there is difficulty in speaking. Later nystagmus, spinal curvature, or some form of talipes may occur and show the cause for this delayed development. The well-marked symptoms are, loss of coördination in the arms and legs, the choreiform ataxia in using the hands, nystagmus, some form of talipes, loss of reflexes, slow, drawling or scanning speech, spinal curvature. There may be headache, vertigo, and slight aching or pains in the limbs. There is no involvement of the sphincters until late in the disease. Atrophy of the muscles is rare except late in the advanced forms of the disease. The electric reactions are generally normal.

Diagnosis.—The affection should be differentiated from **disseminated sclerosis** and **Huntington's chorea**. Distinction from the former is sometimes quite difficult. In the latter the disease generally occurs later in life, there is more involvement

of the mental faculties, there is no spinal curvature, no talipes, and as a rule no nystagmus.

Prognosis.—Unfavorable. The duration of the disease is many years.

Treatment.—There is practically none. Arsenic has been beneficial at times, but only temporarily. Suspension has been tried, but without effect.

QUESTIONS.

What is caisson disease?
What is the etiology of caisson disease?
What is the pathology of caisson disease?
What are the symptoms of caisson disease?
What is the treatment of caisson disease?
What is pressure myelitis?
What is the etiology of pressure myelitis?
What is the pathology of pressure myelitis?
What are the symptoms of pressure myelitis?
What is the diagnosis of pressure myelitis?
What is the prognosis of pressure myelitis?
What is the treatment of pressure myelitis?
Define myelitis.
What are the varieties of myelitis?
What is the etiology of myelitis?
What is the pathology of myelitis?
What are the symptoms of myelitis?
From what diseases must myelitis be differentiated?
Give the diagnosis of myelitis.
What is the prognosis of myelitis?
Give the treatment of myelitis.
What are the synonyms of disseminated sclerosis?
Define disseminated sclerosis.
What is the etiology of disseminated sclerosis
What is the pathology of disseminated sclerosis?
What are the symptoms of disseminated sclerosis?
What is the diagnosis of disseminated sclerosis
What is the prognosis of disseminated sclerosis
What is the treatment of disseminated sclerosis?
What are the synonyms of locomotor ataxia?
What is locomotor ataxia?
What is the etiology of locomotor ataxia?
What is the pathology of locomotor ataxia?
What are the symptoms of locomotor ataxia?
What is the diagnosis of locomotor ataxia?
What is the prognosis of locomotor ataxia
What is the treatment of ocomotor ataxia?
What is the synonym of hereditary ataxia?
What is hereditary ataxia?
What is the etiology of hereditary ataxia?
What is the pathology of hereditary ataxia?

What are the symptoms of hereditary ataxia?
What is the diagnosis of hereditary ataxia?
What is the prognosis of hereditary ataxia?
What is the treatment of hereditary ataxia?

CHAPTER VI.

ORGANIC DISEASES OF THE SPINAL CORD (CONTINUED).

AMYOTROPHIC LATERAL SCLEROSIS.

Definition.—Amyotrophic lateral sclerosis is a nervous disease characterized by a degeneration of the lateral columns and adjacent gray matter, and manifested clinically by the two symptoms of spastic rigidity and muscular atrophy.

Etiology.—The etiology is not definitely understood. Exposure to extreme cold, great physical exertion, and trauma are supposed to act as exciting causes. It is seen oftenest during middle adult life and is more common among males than females.

Pathology.—The pyramidal tracts are symmetrically sclerosed as well as the large cells in the anterior gray cornua. The degeneration may extend as far as the internal capsule. Certain nerve-centres (the hypoglossus and vagus accessory) in the medulla also degenerate, the peripheral nerves also undergo degeneration, which is of the parenchymatous type. In the muscles the essential fibres are replaced by connective tissue and fat, the change in color and consistence being often readily apparent upon quite superficial inspection.

Symptoms.—The affection usually manifests itself in the arm first. Loss of power and wasting, beginning in the small muscles of the hand, gradually extend over the entire body. The muscles become markedly atrophied, especially on the extensor side. The atrophy is not limited to one group of muscles, but seems to occur *en masse*. The reflexes are exaggerated. The power to move the arm is soon totally lost. Electric reaction is normal in the intact muscular fibres, but reaction of degeneration is noticed when the atrophy is

extreme. The arm shows a characteristic paralytic deformity; it lies close to the body; the forearm is semiflexed and pronated; the hand is semiflexed, and the fingers are bent upon the palm. The affected muscles are in a state of marked contracture. Sensation is normal, but the tendon-reflexes are greatly increased. The atrophy and contracture afterward extend to the lower extremities; the gait becomes spastic and paretic, but soon paralysis supervenes. If the bulbar nuclei are involved, there may be wasting of the muscles of the face, with alteration in the expression and impairment of functions as regards speech, respiration, deglutition, and cardiac action. The speech becomes indistinct, swallowing difficult, the tongue atrophied, and general nutrition suffers. Death finally ensues from difficulty in respiration. The functions of the bladder and rectum usually remain normal. In the very late stages of the disease the atrophic symptoms may dominate the picture: the rigidity disappears, the reflexes are lost, and the patient is reduced to a state of bedridden helplessness, but with unimpaired intelligence.

Diagnosis.—Amyotrophic lateral sclerosis is distinguished from **pure progressive muscular atrophy** by the muscular rigidity and exaggerated reflexes.

Prognosis is hopeless as regards cure. Early helplessness is the rule. The disease is fatal within a few years.

Treatment.—This should be symptomatic and palliative. Rest, massage, electricity, and hydrotherapy are all of value. Arsenic and potassium iodide are recommended, but they usually prove useless, except for transitory relief of some of the symptoms.

PROGRESSIVE MUSCULAR ATROPHY OF SPINAL ORIGIN.

Synonyms.—Chronic spinal muscular atrophy; Poliomyelitis anterior chronica.

Definition.—Chronic spinal muscular atrophy is a chronic nervous disease characterized anatomically by degeneration of groups of the motor nuclei in the cord and medulla, and

manifested clinically by a wasting of the corresponding muscles.

Etiology.—The affection is more frequent in males than in females, being usually an affection of adult life. Heredity may play some part in its development. Mental excitement, exposure to wet and cold, injury of the spinal cord, concussion, syphilis, lead-poisoning, and acute infectious disease may act as exciting causes.

Pathology.—The affected portion of the spinal cord (usually the cervical portion is first involved) is softer and smaller than normal in the region of the anterior cornu; the large ganglion-cells are atrophied, or in places have entirely disappeared; the anterior roots and motor fibres of the peripheral nerves and the muscles supplied by them are atrophied. The degeneration of the gray matter may extend to the medulla. The muscles waste and undergo fatty and sclerotic changes.

Symptoms.—The prodromata consist of aching, coldness or numbness, and loss of strength in the affected portion, mostly the upper extremity. The atrophy is first noticed in the small muscles of the thumb; the ball of the thumb becomes flat, and the thumb is in close apposition to the second metacarpal bone. Soon the interossei atrophy, and the palm of the hand is sunken and the fingers assume a "claw-like" appearance from the actions of the extensors. Although one hand is usually affected before the other, the disease tends to become symmetric. Next the muscles of the forearm, arm, and shoulders waste, rendering the bony prominences marked, and the same change takes place in those of the trunk. Those of the neck are rarely implicated. Movements with the arms become difficult, and when the diaphragm or the intercostal muscles become affected respiration is interfered with. The wasting proceeds to an extreme grade, and the patient may be reduced to a mere skeleton. A fibrillar twitching of the affected muscle is noticed early in the disease. The electric excitability may be lost or show a reaction of degeneration. Trophic and vasomotor disturbances may occur. The tendon-reflexes are always absent. The sensation remains nor-

mal, although the patient may complain of numbness and coldness of the affected limb. The functions of the bladder and rectum are not impaired. The legs are not involved until late in the disease, and frequently escape entirely. The face and tongue usually escape the atrophy.

Course.—The process is very slow, and it may be years before the muscles that are supplied from the medulla begin to atrophy. When their nuclei are attacked, all the symptoms of a chronic bulbar paralysis appear, such as indistinct articulation, difficulty in swallowing, and embarrassed respiration.

Complications.—The condition may be associated with amyotrophic lateral sclerosis.

Diagnosis.—It must be distinguished from **primary muscular atrophy**, which usually occurs in younger persons, rarely begins in the hand, and may affect several members of a family.

The **prognosis** is grave, as the disease always ends fatally, though it may be prolonged for very many years.

Treatment.—The disease is incurable. Healthful general surroundings, nutritious food, and massage in some cases may prolong life. When there is a history of syphilis, mercury and potassium iodide should be tried. It is claimed by some authors that hypodermatic injections of strychnine have given good results. Electricity seems to be of no benefit.

PSEUDOHYPERTROPHY OF THE MUSCLES.

Synonyms.—Pseudomuscular hypertrophy; Lipomatous muscular atrophy.

Definition.—Pseudohypertrophy of the muscles is a morbid condition limited to the muscles, and not dependent on a lesion of the central or peripheral nervous system.

Etiology.—It is an affection which develops almost invariably between the ages of five and eight years. (Strümpell.) Many cases show a hereditary predisposition, and frequently several children of the same family are affected. Male children are more liable to be attacked than female. There is found, occasionally, in the affected families some disposition to a nervous taint, such as hysteria, epilepsy, etc. (Strümpell.)

Pathology.—There is no anatomic lesion of the nervous system; it is a primary affection of the muscles. Microscopic examination shows an increase of the interstitial connective tissue, and especially of the fatty tissue between the muscle-fibres. The latter are sometimes atrophied and degenerated.

Symptoms.—The disease is gradual in its onset. The child has difficulty in walking from weakness in the muscles of the back, trunk, and lower extremities. The gait is waddling; the abdomen is very prominent; the spinal column shows a decided forward curve in the lumbar region; the legs are raised with difficulty; and the toes droop. The manner in which a child tries to raise himself from the floor is characteristic: the child usually gets on all-fours first, and then gradually straightens himself by leaning his arms on his knees. (Strümpell.) Sooner or later the movements in the upper extremities become interfered with. The muscular groups show a great increase in volume from superabundant development of fat, but occasionally there is no pseudohypertrophy. Reaction of degeneration is never noticed. The petallar reflex may be diminished or lost. Sensation is normal, and the functions of the bladder and rectum are preserved. The skin, especially in the legs, may show a bluish discoloration. In some cases mental weakness has been noticed.

Prognosis.—The affection advances steadily, the victim of the disease finally becoming bedridden. The condition usually terminates fatally from respiratory disturbances or some other intercurrent disease.

Treatment.—Drugs usually yield no results. Massage, electricity, graduated exercise, and strychnine hypodermatically are recommended with the hope of arresting the progress of the disease.

ERB'S FORM OF JUVENILE OR HEREDITARY MUSCULAR ATROPHY.

Causes.—This affection usually begins in youth, before the age of twenty, and is more common among females than males. It is frequently hereditary.

Symptoms.—In nearly all cases certain sets of muscles atrophy, while others remain perfectly normal. Those mostly attacked are the pectoralis major and minor, trapezius, latissimus dorsi, serratus magnus, rhomboidei, sacrolumbalis, and longissimus dorsi. (Erb.) The muscles of the arm and forearm withstand the effects of the disease for quite a long time. In the lower extremities the glutei, quadriceps, peronei, and tibialis anticus are first to suffer. The functions of the respective parts are soon interfered with, slowly progressing till the loss of function is complete. Sensation, as a rule, is not interfered with, and reaction of degeneration is absent. In some cases the affection begins in the muscles of the face, the so-called **Dejerine-Landouzy type.** It gives the face a typical appearance, due to thickening of the lips, described as the "tapir-mouth." (Sachs.) Examination of the peripheral nerves and the spinal cord has failed to give any evidence of pathologic changes.

The **prognosis** is unfavorable, as the disease slowly but persistently progresses toward complete paralysis and death.

Treatment.—Muscular exercise, electricity, and massage have been recommended and thought by some to stay the progress of the disease. Attention to the general health, tonics, nutritious food, and fresh air, by increasing the vigor of the organism, tend to delay the progress of the degenerative process.

UNILATERAL LESION OF THE SPINAL CORD.

Synonym.—Brown-Séquard's paralysis.

Definition.—A group of symptoms due to a unilateral affection in a part of the spinal cord.

Etiology.—Hemorrhages in the cord, direct injury, bony growths, or callus and tumors sometimes divide or compress a half only of the cord.

Symptoms.—As the sensory fibres of one side of the cord are at every level decussating and passing to the other side of the body, while the motor fibres pass on the same side to the periphery, it is evident that in unilateral lesion there is

loss of motion on one side and loss of sensation on the corresponding side of the body. On the paralyzed side sensation is usually abnormally increased with the exception of the muscular sense, which is diminished; the reflexes are increased, especially the tendon-reflexes, and the temperature is usually higher. On the side opposite to the lesion there are a loss of sensation of touch and temperature, and sometimes a development of bedsores below the level of the lesion. At the level of the lesion there is a band of anæsthesia about the body, with another band of hyperæsthesia above it; the band on the side of the lesion being a little higher than upon the opposite side. (Starr.) Micturition and defecation are disturbed, and there are shooting pains in the affected parts.

Prognosis and **treatment** depend entirely on the primary affection.

QUESTIONS.

What is amyotrophic lateral sclerosis?
What is the etiology of amyotrophic lateral sclerosis?
What is the pathology of amyotrophic lateral sclerosis?
What are the symptoms of amyotrophic lateral sclerosis?
What is the diagnosis of amyotrophic lateral sclerosis?
What is the prognosis of amyotrophic lateral sclerosis?
What is the treatment of amyotrophic lateral sclerosis?
What are the synonyms of progressive muscular atrophy?
What is progressive muscular atrophy?
What is the etiology of progressive muscular atrophy?
What is the pathology of progressive muscular atrophy?
What are the symptoms of progressive muscular atrophy?
What is the diagnosis of progressive muscular atrophy?
What is the prognosis of progressive muscular atrophy?
What is the treatment of progressive muscular atrophy?
What are the synonyms of pseudohypertrophy of the muscles?
What is pseudohypertrophy of the muscles?
What is the etiology of pseudohypertrophy of the muscles?
What is the pathology of pseudohypertrophy of the muscles?
What are the symptoms of pseudohypertrophy of the muscles?
What is the prognosis of pseudohypertrophy of the muscles?
What is the treatment of pseudohypertrophy of the muscles?
What is Erb's form of juvenile muscular atrophy?
What are the symptoms of Erb's form of juvenile muscular atrophy?
What is the prognosis of Erb's form of juvenile muscular atrophy?
What is the treatment of Erb's form of juvenile muscular atrophy?
What is unilateral lesion of the cord?
What is the etiology of unilateral lesion of the cord?
What are the symptoms of unilateral lesion of the cord?
What is the prognosis of unilateral lesion of the cord?
What is the treatment of unilateral lesion of the cord?

CHAPTER VII.

ORGANIC DISEASES OF THE SPINAL CORD (CONTINUED).

PRIMARY LATERAL SCLEROSIS.

Synonyms.—Primary spastic paraplegia; Spastic spinal paralysis.

Definition.—Primary lateral sclerosis is a nervous disease dependent upon a primary sclerotic affection of the lateral pyramidal tracts or columns, and characterized by loss of power, exaggerated reflexes, and a spastic condition of the muscles.

Etiology.—It is a disease of adult life, and is more common in men than in women. It is not very common, and its etiology is not at all definitely known.

Pathology.—The connective-tissue framework which supports the nerve-fibres is increased in thickness. There is a primary degeneration of the axis-cylinder in each nerve-fibre, resulting in its swelling, segmentation, fatty degeneration, and final absorption.

Symptoms are always of gradual onset. The disease may manifest itself at first by a stiffness in walking or in using the arms which gradually increases, and suggests a condition of tonic spasm. The muscles affected do not atrophy rapidly, as their spinal centres are not diseased. Their reflex and mechanical excitability is increased, as the spinal centres are no longer inhibited by brain impulses, so the muscles become hypertrophied from continued reflex stimulation. The symptoms are most objectively conspicuous in the lower limbs. The knee-jerk is exaggerated and ankle-clonus is nearly always present. When put into use the muscles become stiff or spastic, and in walking the gait is peculiar—short, jerky, and spasmodic dragging steps with the knees overlapping. Finally, when paralysis is complete, the legs are drawn up, knees overlapping and so rigid as to be incapable even of passive motion. The muscles show no electric changes. There is imperfect control of the bladder; there

may be involuntary emptying of the bladder at intervals or retention of urine. There is obstinate constipation. Sexual functions may be indirectly lost. There are no sensory symptoms. The upper extremities are not often involved, but finally loss of power and rigidity may develop in them also.

Diagnosis.—The diagnosis is ordinarily a matter of no great difficulty, the clinical picture being very consistent and striking.

Prognosis.—The disease may last many years, the general health remaining quite good. But later, permanent paralysis develops and the patient is incapacitated for any and all forms of physical labor, though the mind is not affected. Recoveries are unknown.

Treatment is symptomatic. Very little may be hoped from measures directed toward a cure. Prolonged rest is essential. For the spasmodic condition of the muscles, hydrotherapy, and the following remedies, bromides, hyoscine, atropine, and conium, will give temporary relief.

ACUTE ANTERIOR POLIOMYELITIS.

Synonyms.—Infantile paralysis; Acute atrophic spinal paralysis; West's morning paralysis.

Definition.—An acute disease occurring almost exclusively in young children, with paralysis followed by rapidly developing atrophy, with degenerative electric reactions in the affected muscles.

Etiology.—Anterior poliomyelitis occurs mostly among children, and generally under three years of age; some authors state that it is more frequent in boys than in girls. It is more common in summer than winter, and seems to show an infectious nature. It is often a sequel to scarlet fever, measles, and diphtheria.

Pathology.—There is an atrophic destruction, more or less complete, of the larger ganglion-cells of the anterior horns. Microscopic examination in recent cases reveals ecchymoses, destruction of ganglion-cells, and infiltration of leucocytes. In later cases an absence or atrophy of the large multipolar

cells in the gray horns, and in their place an increase of connective tissue. The atrophy extends through the anterior nerve-roots to the periphery and thence to the muscles.

FIG. 15.

Infantile paralysis and atrophy of the left arm two years after onset; the partial luxation of the humerus is evident, and also the *main en griffe*. (Starr.)

Symptoms.—As a rule the onset is sudden. Often the child may be put to bed at night in apparent health, and in the morning is found paralyzed in one or more limbs. High

FIG. 16.

Infantile paralysis with atrophy of the right leg. The curvature of the spine is secondary to the shortening of the leg. (Starr.)

fever or chills, general malaise, pain all over the body, decided cerebral symptoms, like delirium or convulsions, and clonic contractions, may usher in the disease. These prodromata may last a short while or several weeks, after which the paralysis is noticed, being extensive as a rule, and affecting one, two, or all the extremities, and sometimes the muscles of the trunk. The general paralysis soon disappears, being left permanently only in one or the other extremity, chiefly in one leg. The cerebral and other symptoms completely disappear, the general condition becomes normal, and the loss of motion in the extremity is the only trace left of the disease. The paralyzed part atrophies rapidly, the reaction of degeneration becomes noticeable after a few weeks, and sometimes the extremity shows a partial arrest of development. The tendon and cutaneous reflexes are absent, sensation is normal, no involvement of the bladder and rectum, and no tendency to bedsores. After a while contractures and subsequent deformities of the extremities appear.

In adults the disease is very rare, but it differs little from that just described. The onset is sudden; convulsions are not so frequent; the paralysis quickly follows, affecting single groups of muscles or a half or the whole of the body. The **subacute** and **chronic forms** differ from the acute form in the less rapid development of paralysis, beginning with weakness, tenderness on pressure, paræsthesia, and then followed by paralysis, which remains stationary for a long time, but may ultimately show signs of recovery.

Diagnosis.—Poliomyelitis is to be distinguished from both **idiopathic muscular atrophy** and **progressive muscular atrophy** by the less abrupt onset of the latter two. The absence of sensory disturbance, paralysis of the bladder and rectum, and bedsores distinguish it from **myelitis.** In **cerebral paralysis of childhood** the presence of cerebral symptoms, of choreiform or athetoid movements in the affected members, and the absence of reaction of degeneration and of early wasting will distinguish it from acute poliomyelitis.

The **prognosis** in the acute stage is somewhat doubtful. The paralysis often improves, and recovery is known to have

occurred, but prognosis in this is unfavorable if the paralysis shows no decided change within the first few months. In adults recovery is more frequent.

The **treatment** during the acute symptoms consists of absolute quiet and rest, a non-stimulating and easily digested diet, ice to the head, counterirritation to the spine, and calomel internally. If there is much fever, the use of baths or such antipyretics as phenacetin and antipyrin. Ergot and sodium salicylate have been employed with success. The affected limb should be wrapped in flannel. After the lapse of two or three weeks the use of electricity should be begun. Both currents should be employed; the galvanic with one electrode over the spine at the level affected, the other on the paralyzed limb. The current strength should be gradually increased as the child becomes accustomed to it. The faradic current should be applied to the limb itself if it induces contraction of the muscles; but if it excites no response, the galvanic current should be used in its place. Massage and gymnastics should be employed along with the electric treatment. At this stage strychnine internally, and gradually increased, is of great value. Splints, braces, and other mechanical appliances will aid in preventing deformity from contractures.

LANDRY'S DISEASE.

Synonyms.—Acute ascending paralysis; Landry's paralysis.

Definition.—Landry's disease is an acute nervous affection characterized by a rapidly progressive motor paralysis beginning in the extremities, usually the legs, and extending upward until it involves the arms and muscles of respiration and of deglutition. It sometimes begins above and progressively descends.

Etiology.—It is a disease of rare occurrence, and its etiology is not well known. It occurs in early or middle adult life, and is more common in males than in females. It is thought by many to be of infectious origin. It sometimes follows the infectious fevers.

Pathology.—No demonstrable lesions have been observed.

Symptoms.—The disease may be ushered in with an elevation of temperature and its associated phenomena, or it may begin with a feeling of extreme weakness, and numbness in the legs. This is progressive, and complete motor paralysis may rapidly follow, involving successively the trunk, upper extremities, and muscles of respiration and deglutition. The sphincters are not involved. In some instances the disease may begin above, and the order of invasion be reversed. The reflexes are abolished. Sensation is usually normal, but there may be hyperalgesia or anæsthesia. The mental faculties usually remain normal. As a rule the muscles do not atrophy or yield the reactions of degeneration. The spleen and lymphatic glands are occasionally found swollen.

Diagnosis.—The **absence** of muscular wasting, reactions of degeneration, and the early involvement of the sphincters in Landry's disease will aid in diagnosing it from **acute myelitis.**

From **multiple neuritis,** in that the sensory disturbances are more marked than in Landry's disease.

The **acute vascular lesions of the cord** may resemble symptomatically Landry's disease.

The **prognosis** is always unfavorable. It usually terminates fatally in the course of a few days, but occasionally the progress of the disease is arrested and recoveries have been reported.

Treatment.—Absolute quiet and rest in bed; hot wet pack to the limbs and cups to the spine are valuable. Ergot, quinine, sodium salicylate, and mercury by inunctions or small, frequently repeated doses, have been recommended. In the protracted cases electricity with potassium iodide and strychnine are indicated.

SYRINGOMYELIA.

Definition.—Syringomyelia is a cavernous condition of the gray matter of the spinal cord associated with an overgrowth of the neuroglia and some degeneration of the surrounding tissue.

Etiology.—It is a rare disease, probably of congenital origin. It is more common among men than among women, and a great percentage of reported cases have been recognized first between the ages of twenty-five and forty years. The exciting causes are trauma, prolonged exposure to cold and dampness, great physical strain, toxæmias, malnutrition, anæmia, syphilis, and alcoholism.

Pathology.—There is a development of gliomatous tissue in the gray matter around the central canal, which later extends outward, involving the entire central gray matter, finally degenerates, and is absorbed, leaving a cavity in the spinal cord. This cavity may extend through the entire length of the cord. Its most frequent situation is in the lower cervical and upper dorsal regions. The disease may invade the posterior horns and columns with symptoms of posterior sclerosis, or it may invade the anterior horns, giving the symptoms of chronic anterior poliomyelitis.

Symptoms.—There is no single pathognomonic symptom, nor is there any constant grouping of symptoms which is positively characteristic of the disease. There is a loss of painful and thermic sensation, while the sensations of touch and location are retained. There are paralysis and atrophy of the muscles, especially of the upper extremities; there may be paralysis of the spinal muscles with curvatures of the spine. If the lumbar region of the cord is involved, the functions of the bladder and rectum are disturbed, and there is loss of sexual power. Vasomotor and trophic disturbances are not infrequent, such as sweating, œdema, cyanosis, blueness of extremities, with lowering of temperature, herpes, bullæ, ulcers, defective growth of the nails. The bones may become quite brittle, causing fractures and joint-affections. The symptoms at first may be limited to one, but later are usually bilateral.

Diagnosis.—Syringomyelia may be confounded with tumor and hemorrhage of the cord, myelitis, and cervical pachymeningitis. In tumor the symptoms are more localized and unilateral, and they progress more rapidly. In hemorrhage of the cord the onset is abrupt and the symptoms are rapidly

destructive. From myelitis and cervical pachymeningitis the diagnosis is at times quite difficult.

Prognosis is bad. The duration of the disease may be many years. Occasionally its progress is spontaneously arrested.

Treatment.—No treatment can arrest the disease. Rest, change of climate, and general tonic treatment (potassium iodide, silver nitrate, and arsenic) are recommended.

ATAXIC PARAPLEGIA.

Synonyms.—Progressive spastic ataxia; Combined postero-lateral sclerosis.

Definition.—Ataxic paraplegia is a nervous disease affecting the posterior and lateral columns of the cord with symptoms of both locomotor ataxia and spastic paraplegia.

Etiology.—It is most common in middle life, and occurs more often in males than in females. The exciting causes are exposure to cold, traumatism, syphilis, and lead and other poisonings.

Pathology.—A sclerosis of the posterior and lateral columns.

Symptoms.—The disease may begin with more or less unsteadiness in standing or walking, and this is more marked in the dark or with the eyes closed. There may be impairment or loss of sexual power, and the sphincters may be affected. There is a gradual and progressive development of paretic weakness in the legs. The reflexes are increased and there may be well-developed ankle-clonus. Rigidity of the legs slowly comes on. Incoördination is well marked. In walking the patient keeps the eyes fixed on the ground and the legs far apart, but the feet are dragged along, and there is not the "stamping" gait with elevation and sudden descent, as is seen in true tabes. Sensory symptoms are rare except aching in the legs and lumbar region. The incoördination may extend to the arms. Trophic symptoms are absent. Mental symptoms may develop late in the disease similar to those of general paresis.

Diagnosis.—Ataxic paraplegia may be diagnosed from **true tabes** by the absence of the patellar reflex, by pupillary changes,

and by sensory symptoms. From **spastic paraplegia** by the absence of ataxia.

Prognosis is unfavorable except in those due to syphilis. The **duration** is many years.

Treatment.—Potassium iodide should be tried in all cases, giving the patient the benefit of the possibility that syphilis may have been the cause.

QUESTIONS.

What is the synonym of primary lateral sclerosis?
What is primary lateral sclerosis?
What is the etiology of lateral sclerosis?
What is the pathology of lateral sclerosis?
What are the symptoms of lateral sclerosis?
What is the diagnosis of lateral sclerosis?
What is the prognosis of lateral sclerosis?
What is the treatment of lateral sclerosis?
What are the synonyms of acute anterior poliomyelitis?
What is acute anterior poliomyelitis?
What is the etiology of acute anterior poliomyelitis?
What is the pathology of acute anterior poliomyelitis?
What are the symptoms of acute anterior poliomyelitis?
What is the diagnosis of acute anterior poliomyelitis?
What is the prognosis of acute anterior poliomyelitis?
What is the treatment of acute anterior poliomyelitis?
What is the synonym of acute ascending spinal paralysis?
What is acute ascending spinal paralysis?
What is the etiology of ascending spinal paralysis?
What is the pathology of ascending spinal paralysis?
What are the symptoms of ascending spinal paralysis?
What is the diagnosis of ascending spinal paralysis?
What is the prognosis of ascending spinal paralysis?
What is the treatment of ascending spinal paralysis?
What is syringomyelia?
What is the etiology of syringomyelia?
What is the pathology of syringomyelia?
What are the symptoms of syringomyelia?
What is the diagnosis of syringomyelia?
What is the prognosis of syringomyelia?
What is the treatment of syringomyelia?
What is the synonym of ataxic paraplegia?
What is ataxic paraplegia?
What is the etiology of ataxic paraplegia?
What is the pathology of ataxic paraplegia?
What are the symptoms of ataxic paraplegia?
What is the diagnosis of ataxic paraplegia?
What is the prognosis of ataxic paraplegia?
What is the treatment of ataxic paraplegia?

CHAPTER VIII.

ORGANIC DISEASES OF THE SPINAL CORD (CONTINUED).

TUMORS OF THE SPINAL CORD.

Sites.—Tumors may appear in the spinal cord or membranes, giving rise to different symptoms according to their seat. These tumors may be extradural or intradural.

All **varieties** may occur. There have been reported lipoma, osteoma, fibroma, sarcoma, myxoma, psammoma, carcinoma, tubercle, gummata, and connective-tissue formations. Cysts or collections of pus may also give symptoms of a morbid new growth. Carcinoma is usually secondary to carcinoma elsewhere. Of the reported cases, sarcoma seems to be the most frequent growing within the spinal canal. The tumor may be single or multiple, and sometimes two varieties are found blended in one tumor. Tubercle, lipoma, and sarcoma are the most common in children. In later life gumma is found to be the most common tumor. Injuries, such as a blow on the spine, have been supposed to be occasional causes. Males are slightly more prone to suffer than females. (Gowers.)

Symptoms.—The symptoms will vary according to the location of the tumor, but nearly all tumors show their presence with the appearance of symptoms of compression, such as shooting pains and motor weakness. In most cases there is anæsthesia on the side opposite to the tumor, while hyperæsthesia exists on the same side as the growth, with ataxia, motor paralysis, and exaggerated reflexes, as the motor and sensory fibres of the cord cross at different levels. There may be rigidity of the spine partly from pain and partly from muscle spasm. Muscular spasm is a common symptom most pronounced when the tumor springs from the membranes. Contractures may develop in the limbs. The paralysis is gradual in its onset; it may be caused by pressure simply or result from myelitis, hemorrhage into the cord, or infiltration of the tumor. The motor paralysis progresses from above downward, while the paralysis of sensation begins at the feet and

ascends. Loss of control over the sphincters usually accompanies motor palsy in the legs. In the later stages bedsores are common and often severe.

Prognosis.—Except in the case of gummata or one that can be removed by operation, the prognosis is very grave.

Treatment.—If the growth is syphilitic, potassium iodide should be given, and in cases of doubt it should be tried. In most cases the treatment is symptomatic, preventing the occurrence of cystitis, bedsores, and relieving pain by sedatives. Many tumors within the spinal canal and outside the substance of the cord may be removed. The early removal of growths within the spinal cord by surgical procedure has resulted favorably.

QUESTIONS.

What are the varieties of tumors of the spinal cord?
What is the etiology of tumors of the spinal cord?
What are the symptoms of tumors of the spinal cord?
What is the prognosis of tumors of the spinal cord?
What is the treatment of tumors of the spinal cord?

PART III.

DISEASES OF THE MEDULLA OBLONGATA.

CHAPTER I.

BULBAR PARALYSES.

PROGRESSIVE BULBAR PARALYSIS.

Synonym.—Glossolabiolaryngeal paralysis.

Definition.—A progressive disintegration of the motor nuclei in the medulla oblongata causing paralysis of the lips, tongue, pharynx, and larynx in the order named.

Etiology.—The etiology is obscure. Heredity seems of slight importance. Cold, emotions, traumatism, and physical exertion may act as exciting causes. It is an affection of adult life, rarely beginning under the fortieth year.

Pathology.—The nuclei of the nerves which supply the atrophied muscles are found under the microscope to have undergone degeneration or to have completely disappeared. The connective tissue is increased; the nerve-fibres, and subsequently the muscles, also atrophy.

The **symptoms** appear gradually, and may be preceded by prodromata of general pain. As a rule the disease is first manifested by an impairment of speech. The difficulty lies in pronouncing the letters that are uttered with the aid of the tongue (alalia). The tongue itself becomes flabby and atrophies progressively, there is a tremor of the tongue and it soon becomes paralyzed. The power of speech is lost, great difficulty in swallowing and chewing appears. With the

tongue the muscles of the lip and face become affected; the labial sounds are pronounced with difficulty. There are atrophy and tremor of the lips with paralysis. There is dribbling of saliva and facial expression is impaired, and the face assumes a thin aspect from atrophy of various muscles of expression. Later the muscles of the pharynx and larynx become paralyzed and deglutition and respiration are interfered with. The food regurgitates or can not be swallowed. It may enter the larynx, producing pneumonia. Cough is impossible. The reflexes as a rule are diminished or absent, but sometimes an increase in the tendon-reflexes of the muscles of the face is met with. Occasionally the muscles of mastication are also attacked by the atrophy. The reactions of degeneration can only exceptionally be demonstrated, as only portions of the affected muscle atrophy. There is no change of sensation or of taste. The pulse is sometimes rapid; temperature from 100° to 103° F. Salivation occurs in most cases, as well as vasomotor disturbances. Occasionally other nuclei may be attacked in the medulla, producing symptoms different from those just given. In some the ocular muscles are affected, while in others both sides of the face are symmetrically attacked. But these forms are rare.

Diagnosis.—Differentiation should be made from slowly developing tumors of the medulla, thrombosis, and hemorrhage in the medulla, and bilateral cerebral affections. The diagnosis is usually readily made.

There is great similarity in the pathogenesis and course of this affection and of **progressive muscular atrophy** and **amyotrophic lateral sclerosis.**

Prognosis is unfavorable. The **duration** of the disease is from two to five years. Death may result from cardiac failure, inanition, or inspiration pneumonia.

Treatment.—It is an incurable affection. Transient improvement may occur by the use of electricity and strychnine. Special care must be taken in feeding these patients, and when deglutition becomes much impaired the stomach-tube should be employed. In other respects the affection is treated symptomatically.

ACUTE BULBAR PARALYSIS.

Synonymn.—Acute bulbar myelitis.

Definition.—An affection characterized by an acute development of marked bulbar paralytic symptoms.

Etiology.—Acute bulbar paralysis may be due to hemorrhagic or embolic softening in the pons and medulla, or to acute inflammatory softening. It is a disease of very rare occurrence.

The **pathology** is probably based on an acute disintegrating inflammation of the medulla.

Symptoms.—There may be **prodromata** of headache and vague pains all over the body, followed as a rule by decided bulbar symptoms. The cases are generally bilateral. The muscles of the tongue and lips are involved and speech is almost or entirely lost. The saliva drools, the lips are flabby and flaccid. There is interference with deglutition, and the laryngeal muscles may be involved, interfering with respiration. There may be elevation of temperature, and the pulse is generally rapid. The extremities are attacked in only very few of the cases.

Prognosis is grave. Death may take place in a few days from interference with respiration.

Treatment.—This disease is incurable. In the beginning of the disease apply counterirritation to the back of the neck. Later in the disease the use of morphine is necessary.

QUESTIONS.

What is the synonym of progressive bulbar paralysis?
What is progressive bulbar paralysis?
What is the etiology of progressive bulbar paralysis?
What is the pathology of progressive bulbar paralysis?
What are the symptoms of progressive bulbar paralysis?
What is the diagnosis of progressive bulbar paralysis?
What is the prognosis of progressive bulbar paralysis?
What is the treatment of progressive bulbar paralysis?
What is acute bulbar paralysis?
What is the etiology of acute bulbar paralysis?
What is the pathology of acute bulbar paralysis?
What are the symptoms of acute bulbar paralysis?
What is the prognosis of acute bulbar paralysis?
What is the treatment of acute bulbar paralysis?

CHAPTER II.

DISTURBANCES OF THE CIRCULATION OF THE MEDULLA AND THE PONS.

HEMORRHAGE INTO THE MEDULLA AND THE PONS.

Etiology and Pathology.—Hemorrhage occurs more often than into the spinal cord, but less often than into the brain. It is probable that preceding the hemorrhage there is always some disease of the bloodvessels, as atheroma or miliary aneurism. The predisposing causes are cardiac disease, nephritis, and alcoholism; the exciting causes are traumatism and local inflammation. The **pathologic appearance** is analogous to that of cerebral hemorrhage.

The **symptoms** usually develop suddenly. There are sometimes slight prodromata of headache and dizziness, but there is generally marked apoplectic seizure. The patient has a shock, falls down, and becomes dizzy or unconscious; or he may have headache, ringing in the ears, vomiting, and clonic spasms. The patient may die immediately if the hemorrhage is extensive. This is probably due to a distinct involvement of the respiratory and circulatory centres. In most cases bulbar paralysis of large or small extent follows. There may be paralysis of the tongue with impairment of speech and difficulty in swallowing. The pharynx, the face, and the extremities may be paralyzed. The paralysis is usually unilateral, the upper and lower extremities on one side being affected, and the face on the other side, because of the decussation of the facial nerve-fibres above the pyramids. But occasionally the hemorrhage may be very extensive and all four of the extremities may be more or less completely paralyzed. There are extremely rare cases of hemorrhage in which the lesion is at the very decussation of the pyramids. This gives the crossed hemiplegia—that is, paralysis of the arm on one side and of the leg on the other. (Strümpell.) Sensation is rarely interfered with, except when the pons is

affected. Vasomotor and respiratory disturbances may also present themselves, and an elevation of the temperature and a rapid irregular pulse are sometimes noticed. Temporary albuminuria and glycosuria are occasionally seen. In fatal cases the body temperature may rise quite high.

Prognosis.—Death may occur almost immediately. The prognosis is favorable if symptoms of absorption present themselves. Occasionally some of the paralytic symptoms persist more or less permanently.

Diagnosis.—To distinguish it from **embolism** is almost impossible. Disturbance of speech and deglutition and the apoplectic onset will diagnose bulbar hemorrhage from hemorrhage elsewhere into the encephalon.

Treatment.—Absolute rest, elevation of the shoulders and head, and ice to the head are advisable. The subsequent symptoms are treated on general therapeutic principles. Potassium iodide, ergot, strychnine, and galvanization are of great benefit.

EMBOLISM AND THROMBOSIS OF THE BASILAR ARTERY.

Etiology.—The medulla and pons derive their chief blood-supply from the basilar artery. An occlusion in any of the branches of this artery may produce a softening in these parts and a subsequent bulbar paralysis. Emboli most frequently follow heart disease and occur in the vertebral arteries, and more often in the left one. Thrombosis is usually a result of a disease of the arteries, as syphilis and atheroma.

Pathology.—The area deprived of the arterial blood undergoes a necrosis and disintegration of tissue and a spot of "softening" results.

The **symptoms** appear very suddenly. There may be an apoplectic attack or a sudden development of paralysis. There may be unconsciousness from the sudden obstruction of the basilar artery causing a disturbance of the circulation in the anterior portion of the brain, and this disturbance of circulation may be to such an extent that the symptoms of

choked disk, rapid pulse, and Cheyne-Stokes' respiration are present. If the patient survive, the subsequent symptoms are those of hemorrhage into the medulla, with decided bulbar symptoms.

Prognosis is generally unfavorable. If the circulation is not restored to normal, death usually ensues after a few days.

Treatment is symptomatic. The same remedies are employed as in other acute bulbar diseases.

QUESTIONS.

What is the etiology of hemorrhage into the medulla and the pons?
What is the pathology of hemorrhage into the medulla and the pons?
What are the symptoms of hemorrhage into the medulla and the pons?
What is the prognosis of hemorrhage into the medulla and the pons?
What is the treatment of hemorrhage into the medulla and the pons?
What is the etiology of embolism and thrombosis in the medulla and the pons?
What is the pathology of embolism and thrombosis in the medulla and the pons?
What are the symptoms of embolism and thrombosis in the medulla and the pons?
What is the prognosis of embolism and thrombosis in the medulla and the pons?
What is the treatment of embolism and thrombosis in the medulla and the pons?

CHAPTER III.

INJURIES OF THE MEDULLA AND THE PONS.

COMPRESSION OF THE MEDULLA.

Etiology.—Injuries, such as fracture or dislocation of the atlas and axis, causing a sudden compression or laceration, may be followed by instant death. Diseases of the bones and structures surrounding the medulla, tumors, and aneurism may produce gradual compression. The mechanical pressure from these either destroys the nervous tracts or interrupts the transmission of nervous influences.

Symptoms.—No exact rules may be formulated as to the manner of manifestation of the different symptoms. The bulbar symptoms are generally more pronounced, such as im-

pairment of speech and deglutition, paralysis of the tongue, soft palate, face, and occasionally motor and sensory symptoms in the extremities are present. Cerebral symptoms, such as vertigo, headache, vomiting, and convulsions, may also appear later in the disease.

The **prognosis** is unfavorable. Death takes place from inspiration-pneumonia or paralysis of respiration.

Treatment is symptomatic and is the same as that of progressive bulbar paralysis.

QUESTIONS.

State the common forms of injury to the medulla and the pons.

What is the usual result of sudden compression or laceration of these parts of the brain?

May gradual compression of the medulla and the pons exist during life? What are its causes? What are its results?

What symptoms supervene upon gradual injury of the medulla and the pons?

Which symptoms predominate in these conditions—bulbar, pontine, or cerebral? What is the usual order of their appearance?

Why would you say the prognosis is essentially grave and what are the usual causes of death in cases of injury to the bulbopontine region?

What is the treatment of bulbopontine injury?

PART IV.

DISEASES OF THE CEREBRUM AND CEREBRAL MEMBRANES.

CHAPTER I.

CEREBRAL LOCALIZATION.

THE LOCALIZATION OF CEREBRAL DISEASE.

Definition.—The localization of cerebral disease is the inference of the situs of an affection from the symptoms it produces.

Functions of the Cerebral Convolutions.—The upper third of the **central convolutions** contains the centres for the movements of the leg of the opposite side. The middle third of the central convolutions contains the centres for the movements of the arm of the opposite side. The upper part of the lower third of the central convolutions contains the centres for the muscles of half the face, and the lower part contains the centres for the lips and tongue.

The **frontal convolutions** contain no motor centres in their upper two-thirds. The lowest frontal convolution on the left side contains the centre for speech.

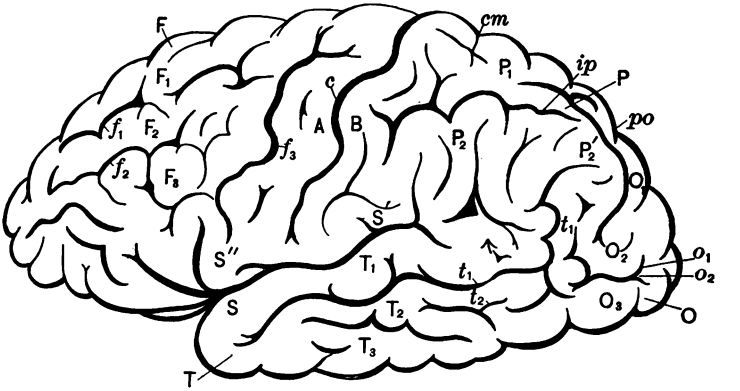
The **parietal convolutions** have no motor centres, but are said to contain the centres for the cutaneous and muscular sensation.

The **occipital convolutions** (especially the cuneus) contain the cortical centre for visual sensations, a lesion here producing hemiopia (only a half of the field of vision being perceived), and occasionally a loss of visual memory.

The **temporal convolutions** (especially the uppermost) contain the centre for hearing of the opposite side. The anterior part of the lobe contains the centre for smell.

The **centrum ovale** contains the fibres of the various cortical centres, consequently injury may cause analogous symptoms as an injury of the cortical portions—hemiplegia, hemianopia, word-deafness, aphasia, and monoplegia.

FIG. 17.



Lateral aspect of the left hemicerebrum: F, frontal lobe; P, parietal lobe; O, occipital lobe; T, temporosphenoidal lobe; S, fissure of Sylvius; S', horizontal, S'', ascending ramus of the same: c, sulcus centralis or fissure of Rolando; A, anterior central or ascending frontal convolution; B, posterior central or ascending parietal convolution; F_1, superior, F_2, middle, and F_3, inferior frontal convolutions; f_1, superior, and f_2, inferior frontal sulcus; f_3, sulcus præcentralis; P_1, superior parietal or posteroparietal lobule; P_2, inferior parietal lobule, viz., P_2, gyrus supramarginalis: P'_2, gyrus angularis: ip, sulcus intraparietalis: cm, termination of the callosomarginal fissure; O_1, first, O_2, second, O_3, third occipital convolutions; po, parietooccipital fissure: o_2, sulcus occipitalis transversus; o_2, sulcus occipitalis longitudinalis inferior; T_1, first, T_2, second, T_3, temporosphenoidal convolutions; t_1, first, t_2, second, temporosphenoidal fissures (lettering according to Ecker).

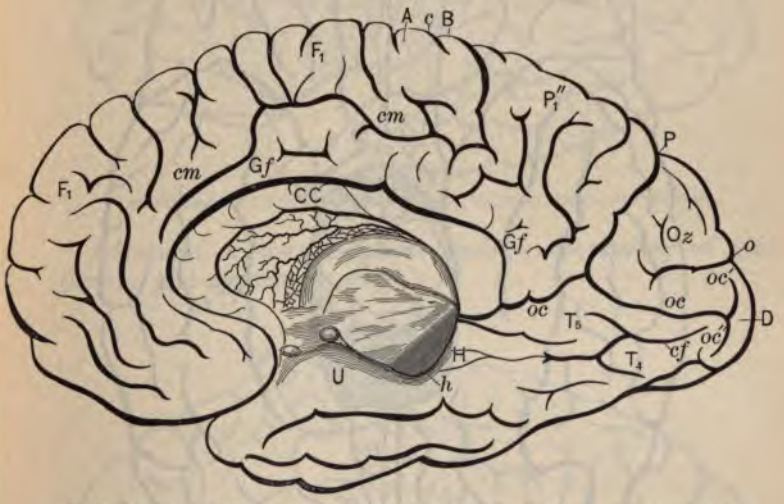
The **central ganglia** (caudate nucleus, lenticular nucleus, and thalamus opticus) when injured produce temporary hemiplegia or hemianæsthesia. The posterior portion of the thalamus also contains the centre for part of the optic nerve.

The **internal capsule** contains in its posterior limb the pyramidal tract, and injury produces complete hemiplegia on

the opposite side of the body. The posterior extremity of the internal capsule contains the sensory tract, and injury produces hemianæsthesia, and sometimes loss of the special senses.

The **anterior pair of the corpora quadrigemina** contain the fibres of the optic nerve, and injury to both causes total blindness.

FIG. 18.



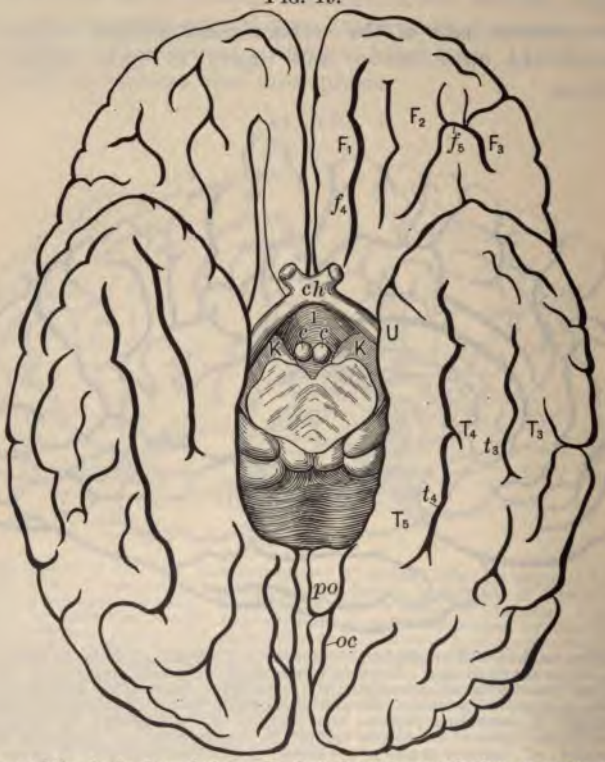
Median surface of the right hemicerebrum: *CC*, corpus callosum cut through the centre; *Gf*, gyrus fornicatus; *H*, gyrus hippocampi; *h*, sulcus hippocampi; *U*, uncinate gyrus; *cm*, sulcus callosomarginalis; F_1, median surface of the first frontal convolution; *c*, terminal portion of the sulcus centralis, fissure of Rolando; *A*, ascending frontal; *B*, ascending parietal convolution; P_2, precuneus; O_2, cuneus; *Po*, parietoöccipital fissure; *o*, sulcus occipitalis transversus; *oc*, calcarine fissure; *oc'*, superior; *oc''*, inferior ramus of the same; *D*, gyrus descendens; T_4, gyrus occipitotemporalis lateralis (lobulus fusiformis); T_5, gyrus occipitotemporalis medialis (lobulus lingualis); *cf*, collateral or occipitotemporal fissure.

The **crura cerebri** contain the pyramidal tracts, the sensory fibres, and the nucleus of the third nerve.

The **pons Varolii** contains the motor fibres of the opposite side of the face, arm, leg, and the nuclei of the fifth, the sixth, and the third nerve of the same side.

The **medulla oblongata** contains the cardiac and respiratory centres, the nuclei of the hypoglossal, spinal accessory, and

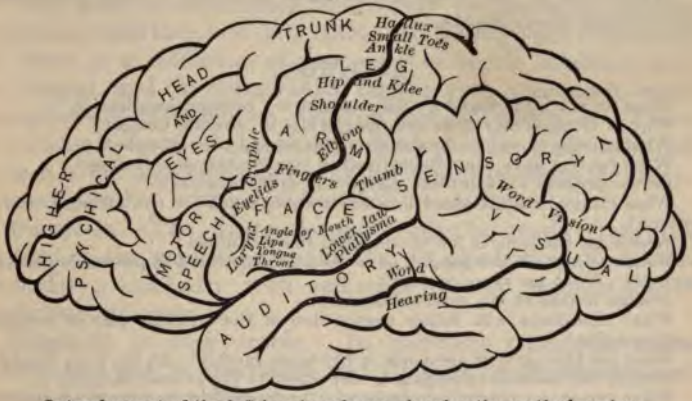
FIG. 19.



View of the brain from below: F_1, first frontal convolution or gyrus rectus; F_2, middle or second frontal convolution; F_3, inferior or third frontal convolution; f_4, sulcus olfactorius; f_5, sulcus orbitalis; T_3, third or inferior temporosphenoidal convolution; T_4, gyrus occipitotemporalis lateralis (lobulus fusiformis); T_5, gyrus occipitotemporalis medialis (lobulus lingualis); t_4, sulcus occipitotemporalis inferior; t_3, sulcus temporosphenoidalis inferior or third temporal fissure; *po*, parieto-occipital fissure; *oc*, calcarine fissure; *U*, gyrus uncinatus; *ch*, optic chiasma; *cc*, corpora albicantia; *KK*, crura cerebri (lettering according to Ecker).

glossopharyngeal nerves, and the motor fibres for the opposite side of the body.

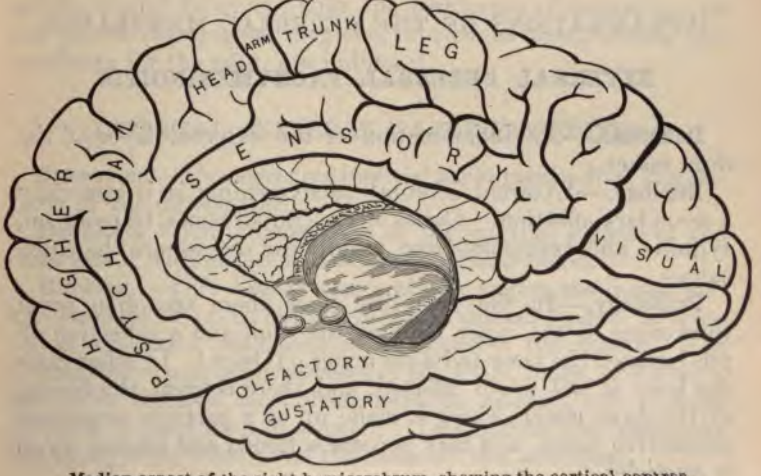
FIG. 20.



Lateral aspect of the left hemicerebrum, showing the cortical centres.

Affections of the **cerebellum** produce uncertainty of gait (ataxia) and a marked vertigo.

FIG. 21.



Median aspect of the right hemicerebrum, showing the cortical centres.

Affections of the **crura ad pontem** produce forced positions and forced movements.

QUESTIONS.

What do you understand by the expression "cerebral localization"?
Why is it important to determine the site of cerebral disease?
What are the functions localized in the central convolutions?
What centres have been determined in the frontal convolutions?
Do the parietal convolutions possess any motor centres?
What clinical conditions follow destructive lesions in the occipital convolutions?
Where are the centres for hearing and smell situated?
Which of the central ganglia have been demonstrated to possess important centres? What are these centres?
Discuss lesions of the internal capsule.
What conditions will follow destruction of the anterior pair of corpora quadrigemina?
What are the nervous elements in the crura cerebri? In the pons Varolii?
Why is the medulla oblongata so important to the animal economy?
What symptoms result from lesions in the cerebellum and crura ad pontem respectively?

CHAPTER II.

INFLAMMATIONS OF THE CEREBRAL MEMBRANES.

EXTERNAL CEREBRAL PACHYMENINGITIS.

Definition.—An inflammation of the external layer of the dura mater.

Etiology.—External cerebral pachymeningitis is generally a secondary affection. Caries of the cranial bones, traumatism, syphilis, and extension from middle-ear disease are the chief causes.

Pathology.—In the syphilitic cases there are often great thickening of the inner table of the skull and a collection of pus between the bone and dura mater. (Osler.) In other cases the bone is not much affected, and there is great thickening of the dura mater, which is made up of a partially organized connective tissue, and may become softened and broken down in particles.

Symptoms.—The symptoms of this affection are indefinite. If it is due to traumatism, there may be headache, nausea, vomiting, delirium, and later coma, and spasms local or general. If there is a collection of pus, signs of septic infection may be present, and the intracranial tension is so great as to cause symptoms of compression with or without paralysis. There is a chronic form of external cerebral pachymeningitis which is seen in old alcoholics and syphilitics, and presents the symptoms of senility with persistent dull headache.

Diagnosis.—The diagnosis may only be made from the history of the associated conditions to which the affection is secondary, especially with reference to any traumatism or syphilitic infection.

Prognosis is unfavorable. In purulent traumatic cases, where the pus is evacuated and drained by trephining, and in those due to syphilis, the prognosis is better.

Treatment will depend upon the associated condition to which the affection is secondary. When it is secondary to traumatism and there is suppuration, the trephine should be used, which may prevent extension of the inflammation to the brain. In cases due to syphilis large doses of potassium iodide should be given, with the bichloride of mercury. In the senile cases, good hygiene, nutritious food, tonics, and anodynes for the pain, are indicated.

INTERNAL CEREBRAL PACHYMENINGITIS.

Synonyms.—Hemorrhagic internal pachymeningitis; Hæmatoma of the dura mater.

Definition.—An inflammation of the internal surface of the dura mater with an accumulation of effused blood.

Etiology.—This disease occurs chiefly among the insane and the subjects of other chronic nervous disorders, and is often found associated with general paralysis of the insane. The process may complicate diseases of the heart, lungs, or kidneys, infectious diseases, alcoholism, syphilis, anæmia, and is more common in men than in women. Traumatism and sunstroke are thought by some to be the cause of this affection.

Pathology.—In milder cases there is usually found a thin encapsulated layer of clotted blood; in chronic cases there are as a rule several successive layers. The collection of blood is found most commonly in the parietal region or at the base of the brain.

Symptoms.—The symptoms will vary according to the extent of the lesion. If the effusion is slight, there are no marked symptoms as a rule. The onset of the hemorrhage is usually sudden in the more pronounced cases: a sharp pain in the head, followed by stupor or coma, a slow pulse, and sometimes inequality of the pupils. The subsequent symptoms depend upon the quantity and location of the effused blood. If in the neighborhood of the motor cortical region, there is paresis or paralysis, or muscular twitching on the opposite half of the body. If the effusion spreads to the other side of the motor region, there may be general paralysis from a compression of the cortical motor centres. In some cases the muscles of half the face may be paralyzed or aphasia is produced. If the effusion be very extensive, death soon follows. Improvement, and even complete recovery, may take place when the blood-clot is absorbed, but it is characteristic of dural hemorrhages that they show a tendency to frequent recurrence. During the intervals between the acute seizures the patient may enjoy good general health, and only in the later stages of the disease show continuous symptoms. Speech is often slow, there is more or less impairment of the mind, and there may be acute maniacal attacks.

Diagnosis.—The diagnosis is not easily made, and the symptoms may be completely obscured by the primary disease.

Prognosis is generally unfavorable. Recovery may occur, but as a rule the disease runs a very protracted course for months or years, sometimes improving and sometimes aggravated. Death as a rule takes place from a general failure of health, or acutely in one of the apoplectic seizures.

Treatment.—The treatment of the apoplectic seizure is absolute rest, constant application of the ice-bag to the head, leeches applied to the temples or mastoid, blisters to the nape of the neck, and the use of full doses of opium and chloral,

unless they are contraindicated by other diseased conditions of the patient. The subsequent treatment is the prolonged use of potassium iodide in small doses. If there is a history of syphilis, it should be given in larger doses combined with mercury. Good hygiene, nutritious food, and tonics are necessary.

ACUTE CEREBRAL LEPTOMENINGITIS.

Synonyms.—Meningitis of the convexity; Purulent meningitis.

Definition.—An acute inflammation of the cerebral pia mater.

Etiology.—Traumatism, surgical operations, sunstroke, and the acute general diseases, including pneumonia, influenza, scarlet fever, ulcerative endocarditis, erysipelas, pyæmia, septicæmia, rheumatism, and typhoid fever, are the predisposing causes. Sclerosis of the bloodvessels, miliary aneurism, and Bright's disease at times influence the development. It occasionally arises from caries of the bone which is secondary to suppurative aural or nasal disease.

Pathology.—The membranes are opaque, thickened, very congested, and adherent, and more or less infiltrated with purulent fluid. The convexity is the part usually attacked, but the base is involved in some of those cases following suppurative disease of the middle ear. The adjacent cortical substance shows œdema and is soft and injected.

Symptoms.—The disease may be ushered in by a chill and a rise in temperature. There is intense headache, which soon becomes violent, and vertigo, delirium, or coma may be added. In addition there may be symptoms referable to an irritation of the cranial nerves, such as nystagmus, trismus, intolerance of light and sound, and fibrillar twitching in the muscles. There is retraction of the head, and occasionally there are also convulsions. The pulse is usually rapid and irregular, and the temperature may reach 104° or 105° F. It runs an irregular course. Vomiting has been noticed in many cases. There is constipation, and the abdomen may be

tense and concave. A diminution in the quantity of urine occurs, and it may show a trace of albumin.

The onset of the disease may sometimes be wholly obscured by the primary affection.

Diagnosis.—Differentiation must be made from typhoid, septic diseases, uræmia, and general tuberculosis. **Typhoid fever** is excluded by its usually slower onset, later appearance of grave cerebral symptoms, the rose-spots, enlarged spleen, the characteristic stools and temperature curve, and the Widal test of the blood. **Septic diseases** are recognized by their history of external wounds, abortions, etc., frequent chills, cutaneous ecchymoses, and septic retinitis. In **uræmia** the character of the urine will sometimes aid in diagnosis. Evidences of **tuberculosis** elsewhere in the body will serve to establish the diagnosis of that disease in the general form.

Prognosis is grave. The affection lasts but a few days, and is nearly always fatal. In a great many cases hyperpyrexia and deep coma precede death.

Treatment.—The patient should be placed in a dark, quiet, well-ventilated room. The head should be shaved if practicable. Constant use of the ice-bag to the head; leeches applied over the mastoid or on the temple; patient should be kept on liquid diet; bowels kept open by use of enemata. For the violent pain and restlessness morpine hypodermatically; if convulsions also occur, chloral and potassium bromide are required. As a prophylactic measure purulent affections of the ear, etc., should be promptly attended to.

CHRONIC CEREBRAL LEPTOMENINGITIS.

Definition.—A chronic inflammation of the pia and arachnoid.

Etiology.—May occur in infancy or childhood from inherited disease or from an acute attack followed by the chronic condition. It is more frequent in male adults and old age. The etiologic factors are traumatism, insanity, antecedent syphilis, tuberculosis, chronic alcoholism, rheumatism, and gout.

Pathology.—The postmortem appearances vary according to the duration and nature of the inflammation. The base is the most frequent seat of involvement in tuberculosis. In simple inflammation there are thickening of bloodvessels and increase of connective tissue in the pia mater. Frequently the cerebral membranes are thickened and are matted together by connective-tissue adhesions, and the dura may be adherent to the skull and the pia to the brain-substance.

Symptoms.—In the cases occurring in childhood there are stupor, headache, occasional vomiting, slight fever, rapid, irregular, or slow pulse, diplopia, strabismus, ptosis, irregularity of the pupils. In some cases there may be convulsions with paresis or paralysis.

The **course** of the disease is very irregular.

Symptoms.—In cases occurring in adults after traumatism the symptoms may be those of any form of chronic mental disease, but the mental symptoms may vary and be irregular. Paresis or paralysis in some form may occur some time during the disease. Constant pain in the head extending over a long period, with mental depression, and the patient giving a history of injury to the head, suggest chronic leptomeningitis at the point of injury. In cases occurring in adults or old age there may be persistent headache, the pain varying in character; rapidly increasing debility; with loss of flesh in the old there may be paresis of the limbs. The state of mind varies from melancholia to exaltation, the latter being more frequent in younger subjects. There may be vertigo or syncope, nausea and vomiting. If the cortex is involved, more decided symptoms appear.

Prognosis is unfavorable. If the disease is recognized early, especially in syphilitics, a cure may result. Operation in focal lesions due to injury sometimes gives good results.

Treatment.—Absolute mental rest, regulated physical exercise, good hygiene, nutritious food, tonics, massage, and electricity are indicated. A change of climate, especially in the summer or in the fall, will be of great benefit. In cases due to syphilis mercury and the iodides should be given to the extent of tolerance where the general health will permit. In

traumatic cases a careful study of the case should be made to decide whether an operation should be done. Counterirritation to the back of the neck may be of benefit.

Ergot and bromides are recommended for the pain. Trional and sulphonal should be used for the insomnia.

TUBERCULOUS MENINGITIS.

Synonyms.—Basilar meningitis; Acute hydrocephalus.

Definition.—Tuberculous meningitis is an acute inflammation of the arachnoidea excited by the tubercle bacillus.

Etiology.—In children the affection may be primary, but in adults it always follows other tuberculous diseases, such as phthisis, tuberculous pleurisy, tuberculous disease of the joints, and tuberculous glands. It is more common in children than in adults, and the majority of cases are seen between the second and fifth years. Heredity, unhygienic surroundings, and poor food are predisposing factors.

Pathology.—The meninges at the base are most involved. The parts about the optic chiasm, the Sylvian fissures, and the interpeduncular space are involved. There may be only slight turbidity and matting of the membranes with serous infiltration; but generally there is a fibrinopurulent exudate, which covers the structures at the base and the adjacent nerves and bloodvessels, and extends out into the Sylvian fissures. The pearly grayish tubercles may usually be seen along the course of the bloodvessels, especially the vessels in the Sylvian fissure, and scattered throughout the membrane. The amount of fluid in the ventricles is increased, and the ependyma is soft and œdematous. The tuberculous deposit at the base is generally seen over the position of the optic, olfactory, and third nerves, and the crura cerebri, and may include any of the cranial nerves. The cortical substance, especially in adults, underlying the affected meninges is also soft and infiltrated with leucocytes.

Symptoms.—The **prodromal signs** of this affection are usually prolonged. For a week or two the child is noticed to be unwell, or it may be convalescent from measles or some other

acute disease of childhood. The disposition of the child changes, it becomes irritable, is restless and peevish, its sleep is disturbed, its appetite impaired, and it loses in weight. In rare cases these prodromata are not observed, and the onset may be acute. Following the prodromal signs the symptoms of the disease may set in suddenly with the signs of **nervous centric irritation,** with a convulsion, or more commonly with headache, vomiting, and fever. (Osler.) The headache becomes intense, and causes from time to time a short sudden cry, the so-called "hydrocephalic cry." The child can not tolerate bright lights or noise of any kind; the surface is also hyperæsthetic, and if touched the patient becomes quite irritable. Constipation is usually present. The fever is slight, but gradually rises to 102° or 103° F. The pulse is at first slow, but **becomes** rapid and irregular later. The pupils are contracted at first. There is muscular twitching, or frequently convulsive seizures which may be local or general. After the irritative symptoms subside the child becomes delirious; there are retraction of the abdomen, obstinate constipation, the pupils are dilated or unequal in size, the head is retracted. The child lies on one side with the limbs drawn up, and the thumb is turned into the palm with the fingers clinched over it. If the finger-nail is drawn across the skin, a red line comes out quickly and remains for some time; this is the **taché cérébrale of Trousseau.**

Later the exudate is sufficient in amount to cause marked pressure-symptoms and to bring on the **stage of paralysis.** Some form of paralysis develops. This may be ptosis, strabismus, amaurosis, facial paralysis, or hemiparesis or hemiplegia. Coma follows delirium; pupils are dilated; pulse very rapid; the respirations may assume the Cheyne-Stokes character (a pause in the breathing, followed by slight inspirations, which grow deeper and deeper, then diminish gradually till the respiration stops again); the temperature falls, and death occurs within one to three weeks, usually after profound paralysis is established.

Diagnosis.—In simple acute meningitis the onset is more abrupt; there is absence of tuberculous foci in other parts or

a tuberculous history, and the range of temperature is usually higher. Tuberculous meningitis must at times be differentiated from **typhoid fever.** In the latter the cerebral symptoms appear later, and the presence of the rose-colored spots, irregular range of temperature, and intestinal symptoms, and the absence of the retracted abdomen and slow pulse in the first stage, will aid in the differential diagnosis of these two diseases.

Prognosis is grave, although some authors claim that recovery is possible.

Treatment.—The patient should be placed in a quiet, well-ventilated, and darkened room, and should have an intelligent, capable nurse. The diet should be liquid and given at regular intervals. An ice-bag should be applied to the head. For the relief of pain, restlessness, and convulsions, chloral, the bromides, and morphine. For the constipation an enema, or the occasional use of a saline purge, which will also relieve the cerebral congestion. If counterirritation is used, the thermo-cautery is the best means. Perchloride of iron, potassium iodide, mercury, and ergot are recommended by some authors. Quincke's lumbar puncture has been used with success in some cases. A few surgeons have trephined and washed out the exudate with antiseptic solutions.

QUESTIONS.

What is external pachymeningitis?
What is the etiology of external pachymeningitis?
What is the pathology of external pachymeningitis?
What are the symptoms of external pachymeningitis?
What is the diagnosis of external pachymeningitis?
What is the prognosis of external pachymeningitis?
What is the treatment of external pachymeningitis?
What is internal pachymeningitis?
What is the etiology of internal pachymeningitis?
What is the pathology of internal pachymeningitis?
What are the symptoms of internal pachymeningitis?
What is the diagnosis of internal pachymeningitis?
What is the prognosis of internal pachymeningitis?
What is the treatment of internal pachymeningitis?
What is chronic leptomeningitis?
What is the etiology of chronic leptomeningitis?
What is the pathology of chronic leptomeningitis?
What are the symptoms of chronic leptomeningitis?

CHAPTER III.

DISORDERS OF THE CEREBRAL VASCULAR SYSTEM.

THROMBOSIS OF THE CEREBRAL SINUSES.

Etiology and Pathology.—Thrombosis in the cerebral sinuses is always a grave affection. The chief predisposing causes are phthisis, cancer, general marasmus, and acute specific diseases. The exciting causes are suppurating disease of the cranial bones, erysipelas, and mastoid abscess. The superior longitudinal sinus is the one mostly affected in thrombosis due to marasmus. That due to extension of inflammation from some neighboring part affects usually the petrosal, transverse, or cavernous sinuses. The thrombus, when extensive, may cause hyperæmia in the meningeal and cerebral veins, and subsequent extravasation of blood.

Symptoms.—When slight, the symptoms are not noticeable. When severe and occurring in children, there are usually coma, rigidity of the muscles of the neck and back, strabismus, nystagmus, and occasionally clonic spasm of the muscles of the face and limbs. In adults there may be prodromata of headache, drowsiness, delirium or coma, and visual disturbances, followed by paralysis in the distribution of the

cranial nerves and in the extremities. If the thrombus suppurates, there are symptoms of septic infection. The paralysis may be permanent or transient, depending on the absorption or elimination of the clot.

The **prognosis** is generally unfavorable, especially where there is a suppurative phlebitis.

The **treatment** is symptomatic.

CEREBRAL HEMORRHAGE.

Synonym.—Cerebral apoplexy.

Etiology.—Hemorrhage into the brain-substance is almost always due to an affection of the walls of the large or small cerebral arteries (miliary aneurism), producing rupture and subsequent hemorrhage. A vein is occasionally affected. The aneurism may affect the larger or the minuter arteries, and the miliary aneurisms are always abundant in number. The affection is most commonly met with in the aged—that is, in persons over fifty years of age—and is more common in men than in women. All causes which lead to degeneration of the arteries, such as nephritis, rheumatism, syphilis, gout, and alcoholism, predispose to it. Of these, nephritis is one of the most certain causes, on account of the associated arteriosclerosis and cardiac hypertrophy and later degeneration of the bloodvessels. Heredity also predisposes to it. Purpura, pernicious anæmia, leucocythæmia, septicæmia, severe infectious diseases, and traumatism may also be productive of cerebral hemorrhage. Some of the immediate provoking causes, by increasing the blood-pressure, are: straining at stool; lifting heavy weights; violent mental excitement, as rage, fright, etc.; and some reflex disturbance, as gastric irritation. Paroxysms of whooping-cough or convulsions may cause it in children.

Pathology.—Hemorrhage is most frequently met with in the corpus striatum, from rupture of one or the other middle cerebral artery or one of its branches, and hardly ever occurs in the cortex except in children. The size of the clot varies; sometimes it is small, merely a capillary oozing, or it may

fill a hemisphere. When the hemorrhage is extensive, the surrounding parts are compressed. The effused mass is surrounded by a wall of torn cerebral tissue, and the blood-clots are mixed with broken-down nerve-tissue and fat-cells. The parts surrounding the clot are softened. The clot is sometimes absorbed, and leaves in its place a cyst filled with serum. Occasionally the absorption is complete, and only a scar is left to indicate the seat of lesion. Sometimes in large effusions in the motor path such secondary changes may be produced as a softening of the cerebral tissues beyond, or a degeneration which travels down the lateral column of the cord on the side opposite the lesion. The bloodvessels are usually found in an atheromatous condition, and are sometimes the seat of miliary aneurisms.

Symptoms.—In a great many cases there are **prodromal symptoms** of headache, dizziness, pallor or flushing of the face, fulness in the head, flickering before the eyes, ringing in the ears, disturbed sleep, inattention, and imperfect memory; temporary attacks of numbness or peculiar tingling in one-half of the body. When the hemorrhage takes place, there is usually loss of consciousness. The nearer the hemorrhage to the cortex, the more pronounced are the symptoms.

In the attack, if the hemorrhage is extensive, the patient falls suddenly into deep coma, which may soon prove fatal. If the hemorrhage is slight at first and gradually increases, the symptoms correspondingly grow worse: the patient at first is delirious, then one arm, one side, and finally the whole body, become paralyzed, and unconsciousness, even death, may ensue from paralysis of the cardiac and respiratory centres. In many cases the patient falls unconscious without previous warning. The face is red, the eyes are injected, the lips are blue, the pulse is full and slow, the respirations are slow and deep; the temperature is at first subnormal from shock, but later it is elevated from irritation. The head and eyes may be strongly rotated to one side, generally toward the injured side. The pupils may be unequal. The paralysis may not be noticeable at first during the comatose state, as the patient lies perfectly motionless, but in some instances

there is a tonic rigidity of the muscles when the hemorrhage is located in the lateral ventricle, or there are epileptiform convulsions when the hemorrhage occurs near the cortical motor centres. The urine and feces may be passed involuntarily. The urine may be retained and usually contains albumin and sugar. Sensation seems to be retained to a greater or less degree. In some cases which are very grave the patient does not awake from the coma, the pulse becomes very feeble, the respirations of the Cheyne-Stokes type, the reflexes are lost, mucus collects in the throat, there is an elevation of temperature, and death may occur in a few hours or in a day or two. In other cases the clot in the brain is gradually absorbed, and there is a slow return to consciousness. Occasionally relapses from renewed hemorrhages occur. In mild cases, instead of the deep coma there are only headache, faintness, nausea, and vomiting. The injury resulting from cerebral hemorrhage depends on the location of the clot. In some cases the resultant symptoms are only temporary if the blood-clots become absorbed. In other cases the injury is permanent, as a destruction of brain-tissue from constant pressure of the clot may follow.

Subsequent Symptoms.—When the attack does not prove fatal, consciousness is restored and there remains a hemiplegia on the side opposite to that of the seat of the injury. After a few hours the affected muscles become rigid from irritation of the motor fibres. This primary rigidity may last from a few days to several weeks. The paralysis is generally not a complete hemiplegia; the muscles of the trunk and respiration are very rarely affected. One side of the face appears flatter than the other; the angle of the mouth on the well side is drawn over; the eyelids close imperfectly on the paralyzed side; the tongue when protruded deviates toward the paralyzed side and articulation may not be complete; the soft palate appears more flabby on the paralyzed side, and the uvula deviates to one side or the other; the shoulder on the paralyzed side is lower than on the sound side. The upper and the lower extremity on the paralyzed side are totally immovable; or only certain groups of muscles are affected, or the

affected limbs are simply paretic. The deep reflexes are exaggerated on the affected side, and the skin-reflexes are diminished. Sensation is unimpaired except when the internal capsule is involved, in which case there is hemianæsthesia. Aphasia of some kind often accompanies right hemiplegia. The gait is peculiar: in walking the patient supports the paralyzed arm; the leg is stiff and extended, does not bend easily at the knee, and the foot is dragged on its inner edge and swung around in walking. In many cases the paralyzed parts gradually regain their functions within a few weeks, but restoration is not always complete. The power of the facial muscles is usually restored entirely, and the leg improves more than the arm. The improvement gradually goes on for several months, but later the process seems to come to a standstill and the paralyzed muscles become contracted. This is the secondary rigidity, and indicates the existence of descending degeneration in the motor tract. Occasionally involuntary movements have been observed in the paralyzed parts (posthemiplegic chorea). Reaction to electricity is usually normal. When the disease is of long duration, trophic and vasomotor disturbances and swelling of the joints may occur. The subsequent mental condition often shows impairment of intelligence, memory, and mental grasp. There may be a lack of control of the emotions, so that the patient laughs or cries with little cause or may show an irritability of temper.

Diagnosis.—The differentiation of the coma of apoplexy from that of **uræmia, opium-poisoning, alcoholism,** and **epilepsy** is frequently quite difficult. The previous history and mode of onset may aid in diagnosis, such as history of constant drinking in **alcoholic coma**; convulsions having preceded coma in **epilepsy**; and the development of coma being more gradual in **opium-poisoning.** The differential diagnosis between cerebral hemorrhage, cerebral embolism, and cerebral thrombosis is never positive.

Embolism.—This may occur at any age, but usually in earlier life, and associated with heart disease. Its onset is sudden and there may be no loss of consciousness. It has less

disturbance of temperature. There is also a more marked improvement of symptoms within twenty-four hours.

Thrombosis may occur at any age, but usually in middle-aged men and associated with a syphilitic history. The onset is more gradual without coma. Temperature does not become subnormal. Consciousness, if lost, returns more quickly than in hemorrhage.

Tumors and Abscess in the Brain.—The hemiplegia develops more slowly, and there is a history of constant headache, vomiting, vertigo, and certain ocular phenomena.

Prognosis.—When there are deep coma, embarrassed respirations, vomiting, prolonged semiconsciousness, and extension and complete paralysis, the prognosis as to immediate danger to life is grave; and if life is prolonged, the outlook is very unfavorable as to the chance of functional recovery. In all cases one should be guarded in giving a prognosis as to ultimate favorable recovery as the patient is liable to subsequent attacks.

Treatment.—*Prophylaxis.*—Patients predisposed to apoplexy, or if there are any suspicions of prodromata, should lead a quiet life, and be warned against any physical or mental exertion; the bowels should be kept open by the occasional use of a saline laxative; high-tension pulse may be eased by mild depressants. They should be given easily digested diet, should be kept in a warm atmosphere, and protected from chilling, and directed to avoid heart stimulants.

During the Attack.—The head and shoulders should be elevated and an ice-bag applied to the head; some writers recommend that the ice-bag be placed over the carotids. A drop of croton oil in glycerin or olive oil should be placed on the back of the tongue to cause quick catharsis. If there is a high-tension pulse with a regular, strong heart action, venesection is indicated. The cardiac depressants, such as gelsemium, veratrum, or aconite, are recommended by some where the pulse warrants their use. Stimulants should be avoided except where heart failure is present. Constriction of the extremities near the trunk is used by some for autodepletion. Warm bottles should be placed to the extremities. If the bladder is full, it

should be catheterized. Try to prevent the formation of bedsores.

Subsequent Treatment.—Potassium iodide (gr. v–x three times a day) should be given to favor quick absorption of the clot. When the rigidity of the muscles has subsided, the use of electricity, massage, and passive movements to the affected muscles should be begun, to keep them healthy. The use of strychnine by the mouth or injected directly into the muscle is recommended. Pay special attention to the care of the general health.

CEREBRAL EMBOLISM AND THROMBOSIS.

Etiology.—A plug may be carried by the arterial circulation into the brain, where it is arrested by an artery smaller than the plug (embolism), or a clot may form within the cerebral artery at a certain spot (thrombosis).

Cerebral embolism may be derived from the vegetations of a fresh endocarditis or of a recurring endocarditis; more frequently from the latter; from a clot in an aneurism or in the heart; from thrombi in atheroma of the aorta or the large vessels. It may occur in a general septic disease, and in the puerperal condition. Embolism may occur at any age with heart disease, or after childbirth, and is more frequent in females than in males.

Thrombus arises from a local disease of the wall of a cerebral artery, causing a roughening of its surface and a subsequent deposition of fibrin. Severe constitutional illnesses, like typhoid fever and cancer, syphilitic endarteritis, and arteriosclerosis, are productive of thrombosis, which in its turn may cause embolism, or *vice versâ*. A thrombus may occur at any age, but generally in middle adult life and in syphilitic persons.

Pathology.—If an embolus or thrombus occurs, the circulation is often reëstablished in the affected part of the brain through the formation of collateral branches; but if this fails to take place, the tissues are deprived of their blood-supply and are transformed into a soft mass, which often assumes a

reddish or yellowish tinge from the blood-corpuscles present. This degeneration takes place within a few days, and if collateral circulation be established by that time, the nerve-tissue may be restored; but if not, the destruction is permanent. The soft mass may be absorbed, leaving a cavity or a scar, and in some cases a cyst with smooth walls and serous contents. In **embolism** the artery most commonly affected is a branch of the left middle cerebral. In **thrombosis** the middle cerebral, basilar, and vertebral arteries are most commonly affected. If the embolism is of septic origin, the local affection in the cerebral artery may be followed by a suppurative disease of the brain.

Symptoms of Embolism.—The onset is usually sudden without any prodromata; if in the middle cerebral artery, there is hemiplegia and sometimes aphasia, but generally no loss of consciousness; or if so, it returns rapidly. The temperature does not drop, but may show a slight elevation. If the basilar artery is blocked, there may be bilateral paralysis from involvement of both motor paths, and later bulbar symptoms may be present, as paralysis of the lips, pharynx, and œsophagus, disturbed heart action, and Cheyne-Stokes breathing.

Symptoms of Thrombosis.—Generally slow in its onset, thrombosis usually has premonitions. Coma is not frequent, but there is usually dulness of mind. In their further course the symptoms of thrombosis are like those of embolism.

Subsequent Symptoms.—In both embolism and thrombosis if softening occurs, the symptoms are identical with those following cerebral hemorrhage. Cerebral hemiplegia, with or without aphasia, is the most common sequela of thrombosis or embolism, as the middle cerebral artery supplying the internal capsule is generally the seat of affection.

Diagnosis.—Embolism and **hemorrhage** greatly resemble each other, and frequently it may be impossible to distinguish between the two conditions.

In **embolism** the occurrence is more common in young people than in old; there is a history of valvular disease; premonitions rarely occur; loss of consciousness is not so frequent as in hemorrhage; temperature does not fall, but may rise to 102° F.

Prognosis is the same as in cerebral hemorrhage. In thrombosis where syphilis is the cause there is more likelihood of recovery.

Treatment.—In embolism absolute quiet is necessary for several days and the after-treatment for paralysis is the same as in hemorrhage. In thrombosis when syphilis is the cause large doses of potassium iodide should be given, and if the syphilis has been recent, mercurial inunctions should be employed at the same time. In other cases the treatment is of no use.

QUESTIONS.

What is the etiology of thrombosis of the cerebral sinuses?
What is the pathology of thrombosis of the cerebral sinuses?
What are the symptoms of thrombosis of the cerebral sinuses?
What is the prognosis of thrombosis of the cerebral sinuses?
What is the treatment of thrombosis of the cerebral sinuses?
What is the etiology of cerebral hemorrhage?
What is the pathology of cerebral hemorrhage?
What are the symptoms of cerebral hemorrhage?
What is the diagnosis of cerebral hemorrhage?
What is the prognosis of cerebral hemorrhage?
What is the treatment of cerebral hemorrhage?
What is the etiology of embolism and thrombosis?
What is the pathology of embolism and thrombosis?
What are the symptoms of embolism and thrombosis?
What is the diagnosis of embolism and thrombosis?
What is the prognosis of embolism and thrombosis?
What is the treatment of embolism and thrombosis?

CHAPTER IV.

INFLAMMATION AND TUMORS OF THE CEREBRAL SUBSTANCE.

ABSCESS OF THE BRAIN.

Synonym.—Suppurative encephalitis.

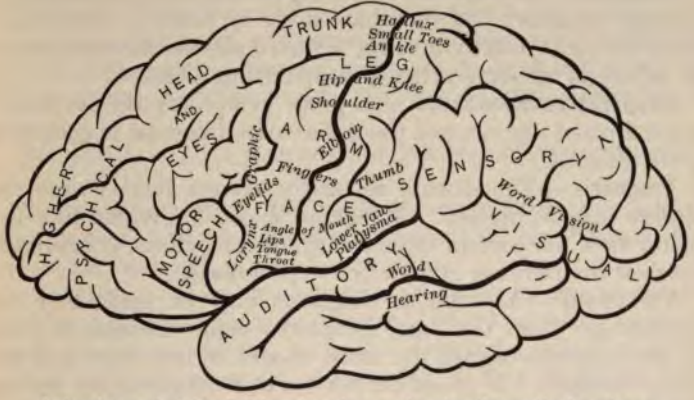
Etiology.—Abscess of the brain is always a secondary condition caused by entrance into the brain of microörganisms from some adjacent or remote infected area in the body. This disease is more frequent in adolescence and middle adult life,

and is more common in males than females. The most frequent source of infection is chronic otitis media, and the next most usual cause is fracture of the cranial bones. It may be secondary to caries of any of the cranial or facial bones. It may be secondary to pus-accumulations anywhere in the body, as in pulmonary abscess, hepatic abscess, pyosalpinx, and ulcerative endocarditis. It may follow the acute infectious diseases. Pyogenic diseases of the nose and throat are sometimes the source of infection. Occasionally it seems to be of idiopathic origin, in which case it is probably due to the entrance of a septic microörganism.

Pathology.—The abscess may be large or small. The temporosphenoidal lobe, the frontal lobe, and the cerebellum are the most frequent seats. The cerebral tissue surrounding the abscess is usually hyperæmic, œdematous, and more or less infiltrated. In the acute cases the abscess may extend rapidly, not becoming encapsulated, and cause death. In the chronic form a thick fibrous sac forms around the pus, preventing it from encroaching upon the neighboring structures; this is called the encapsulated variety. The pus is of a greenish color, and mixed with broken-down nerve-tissue. Occasionally multiple abscesses develop in the brain secondary to distant foci of suppuration.

Symptoms.—The symptoms may develop rapidly or they may be of gradual onset. After an injury cerebral symptoms may develop suddenly, such as intense headache, delirium, vomiting, mental distress, rigors, a high temperature, and sometimes convulsions and coma. In chronic cases the headache is not so intense as in the acute form; vertigo may be slight; vomiting rare; there are irritability and mental impairment; the temperature is irregular; there is stupor, and the patient loses flesh and strength. According to the location of the abscess, there may be different focal symptoms. If the motor area is involved, there may be convulsions or paralysis in one limb. If the temporosphenoidal lobe is involved, there may be deafness or aphasia; if the occipital lobe, there is hemianopia; if the cerebellum, there is a cerebellar staggering.

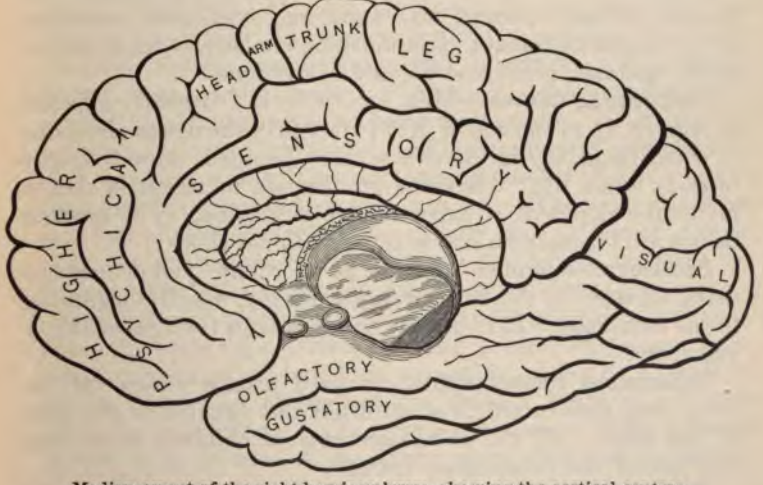
FIG. 22.



Lateral aspect of the left hemicerebrum, showing the cortical centres.

The **duration** of the affection may be weeks or months, and occasionally even years.

FIG. 23.



Median aspect of the right hemicerebrum, showing the cortical centres.

Diagnosis.—Cerebral tumor greatly resembles in its symptoms cerebral abscess; but in tumor fever is usually absent, and choked disk is usually present, while in abscess there is history of injury or of some primary suppurating disease.

Suppurative meningitis also gives symptoms similar to the acute form of abscess, and the differential diagnosis is generally quite difficult.

Prognosis.—The disease is nearly always fatal unless the abscess may be opened. When an accessible region like the motor area, temporosphenoidal lobe, or cerebellum, is involved, surgical interference gives a better prognosis.

Treatment.—As soon as diagnosis may be made and the localization from the focal symptoms shows the abscess to be in an accessible region, the skull should be trephined and the pus evacuated. If operation can not be resorted to, an ice-bag should be applied to the head, and narcotics used to relieve the distress.

TUMORS OF THE BRAIN.

Varieties (in order of frequency).—Gumma, tuberculous tumors, glioma, sarcoma, carcinoma, and occasional varieties, as fibromata, osteomata, psammomata, cholesteatomata, angiomata, lipomata, echinococcus, and cysticercus.

Predisposing Causes.—Men are twice as frequently affected as women up to the age of fifty; after fifty the disease is about of equal frequency in the two sexes. Tumor is more frequent in early adult life. Heredity predisposes to tumor in that it favors development of tubercle and carcinoma. The **exciting causes** are traumatism and severe emotional shock.

Pathology.—Gumma appears as a round yellow caseous mass, situated on the base or in the cortex, usually beginning in the meninges. It is usually seen between the ages of thirty and forty.

Tuberculous tumors appear as hard masses which vary in size; they may be single or multiple and situated in any part of the brain. Of the cases occurring in children, more than half are of the tuberculous variety.

Glioma arises from the neuroglia. It may appear firm and

hard, or soft and quite vascular; usually situated in the white substance. It is observed more frequently in the young.

Sarcoma may be of any variety, as fibrosarcoma, round-cell, spindle-cell, and gliosarcoma, and occurs most commonly in the membranes of the brain. It may be very diffuse in its growth.

Carcinoma is quite rare and is often secondary to cancer in other parts.

Cysts are frequently the result of hemorrhage or softening, and appear between the membranes and the brain. The **porencephalous cyst** is usually congenital.

Symptoms.—Most new growths originate in the meninges, and by compressing a certain part of the brain they produce their special symptoms, such as: Headache, which may be severe and persistent or paroxysmal; it may be diffuse over the entire head, but occasionally the pain may be localized and associated with tenderness on pressure. Vomiting is a common feature, especially in tumors in the posterior fossa, and it may occur with or without nausea or relation to food. Optic neuritis or choked disk occurs in four-fifths of all the cases. (Gowers.) It may be found in only one eye, but usually in both. The other ocular phenomena are optic atrophy, diplopia, hemianopia, and blindness. The mental disturbances are impairment of memory, dulness and apathy, irritability of temper, and sometimes dementia. There is often vertigo or sense of giddiness. The patient may complain of this on rising suddenly or on turning quickly. The pulse is slow and irregular (between 50 and 60). There are insomnia and loss of flesh and strength. There may be loss of control over the sphincters, especially when the tumor involves the frontal lobes. In tumors in the posterior fossa there may be glycosuria and polyuria. There may be general convulsions of an epileptic type. Besides these general symptoms, cerebral tumors also produce local symptoms, either as a result of direct destruction of brain-tissue or as a result of pressure upon the brain-tissue. The most common seat of the different tumors is at the base of the brain, where they cause compression of the various nerve-trunks and subsequent paralysis of

the muscles supplied by these nerves. The paralyzed muscles always give the reaction of degeneration, showing that the paralysis is peripheral and not central. When the tumors are situated in the cerebral hemispheres, hemiplegia usually develops, but always very slowly. In general the focal symptoms always depend upon the exact location of the tumor, and accordingly there may be different focal symptoms, which were described in the section on Localization. As to the nature of the cerebral tumor, those most commonly found are gumma, tuberculous tumors, or glioma. A general tuberculous tendency or a syphilitic history points to a tuberculous or syphilitic tumor. In young people with no syphilitic history the new growth is likely to be tuberculous; in adults with no phthisical history the new growth is likely to be syphilitic. If the tumor follows a carcinoma or sarcoma elsewhere in the body, it is most likely of a malignant nature.

Diagnosis.—From **abscess**, by history of injury or suppurative disease of the middle ear; by severity of early symptoms in abscess; optic neuritis is rare in abscess and there is often febrile disturbance.

From **chronic nephritis**. The latter disease occurs more frequently in older persons than tumor; the headache is not so severe as in tumor. In chronic nephritis examination of the urine and condition of the heart, arteries, and blood-pressure will help to differentiate the two.

Hysteria.—Cerebral tumor must be distinguished from hysteria, especially with migraine, by a study of the patient and separation of the subjective or imaginary from the objective symptoms and by examination of the condition of the optic disk.

From the general symptoms of headache, optic neuritis, vomiting, and the local symptoms of pressure, the existence of tumor may be determined. The **variety of tumor** can not always be diagnosed, but the history and the probable frequency of various tumors at different ages will be of great aid.

Prognosis.—The tuberculous growths in children and syphilitic tumors in adults give the most favorable prognosis.

Occasionally a sarcoma may become capsulated and cease to grow or even decrease in size. The duration of the disease is from a few months to three years, and in exceptional cases life is prolonged for many years.

Treatment.—In **cerebral gumma** potassium iodide and mercury should be given. The potassium salt should be given in increasing doses and pushed to the point of tolerance, and at the same time inunctions of mercury should be employed. In the early course of **any cerebral tumor** when syphilis can not be excluded, the patient should have the benefit of the doubt and be given the antisyphilitic treatment for at least six weeks, and then if the symptoms do not begin to yield, one may exclude syphilis. In both **tuberculous** and **syphilitic growths** the patient should be given a nutritious diet and general tonics, such as codliver oil, iron, arsenic, and quinine. The bowels should be kept open and special attention paid to the digestive organs.

For the headache an application of the ice-cap to the head, mustard plaster to the nape of the neck, or the application of the Paquelin cautery may be tried. The drugs recommended are, cannabis Indica, some of the coal-tar products, codeine, and as a last resort morphine. For the vomiting mustard should also be applied over the stomach. For the convulsions chloral hydrate by the rectum and morphine hypodermatically. If syphilis has been excluded, and certain tumors as fibromata, etc., are located in an accessible position, surgical interference may be of value.

CEREBRAL SYPHILIS.

Etiology.—The symptoms of cerebral syphilis belong to the tertiary stage of the disease, and are rarely ever observed until at least one year or longer from the date of the primary lesion. The interval may be ten or twenty years. Both sexes are equally liable to be attacked by the disease, and it may occur at any age. Hereditary syphilis sometimes produces the symptoms of cerebral atrophy or the symptoms of disseminated cerebrospinal sclerosis in children.

Pathology.—Syphilis may produce a circumscribed tumor, a disease of the arteries, or a general sclerotic infiltration of the brain. The circumscribed tumors are small, yellowish in color, cheesy in the centre, and originating in the dura mater spread to the brain-tissue. The arterial disease causes a thickening of the bloodvessels, a narrowing of their lumina, and thus produces thrombosis. The arteries most frequently involved are those at the base of the brain, especially the middle cerebral artery and its branches.

Symptoms.—The **symptoms of gumma** at the base of the brain are persistent headache, worse at night, insomnia, mental depression, impairment of memory, vertigo, distressing sensations in the head, sometimes vomiting, and local symptoms of paralysis of the nerves at the base of the brain—the third and sixth suffer the most frequently. If the exudate is mainly upon the convexity of the brain or over the cerebellum, the symptoms are spasms and paralysis, paræsthesia and anæsthesia, hemiopia, or some form of aphasia. With these cases violent epileptiform convulsions may appear.

The **symptoms of cerebral gumma** are very much like those of tumor of the brain, but the former is usually more rapid in its onset.

Symptoms of Syphilitic Endarteritis.—This is a common variety of syphilis of the brain. This causes such a diminution in the calibre of the bloodvessels that there is an anæmia of that part of the brain supplied by the vessels involved, which finally produces thrombosis and softening in that part of the brain cut off from its blood-supply. In this affection there is frequently a prodromal stage, such as temporary aphasia, numbness or weakness in one limb or in one-half of the body, disturbance of vision or vertigo; then when thrombosis occurs, which is frequently quite suddenly, the symptoms of cerebral apoplexy appear. The intensity of the initial shock varies; occasionally there is only a slight dizziness; again there is a coma that lasts for days. Mental condition and dulness, which may persist for weeks, sometimes follow the shock. After such attacks there is usually a spot of softening in the brain, and complete recovery is rare.

Symptoms of Diffuse Cerebral Syphilis.—When syphilis is diffuse, it resembles multiple sclerosis in its symptoms. The memory and speech become impaired by degrees, the motor powers are lost, and after a protracted illness the patient finally dies.

Diagnosis.—**Tumors, cerebral softening, hemorrhage, multiple sclerosis, and paralysis of the insane** greatly resemble cerebral syphilis. The most important point in diagnosis is the etiology of previous infection with syphilis, which is determined by the history of the patient and the objective signs on other parts of the body, such as enlarged glands, ulcers, tibial periostitis, and scars on skin or mucous membrane. An apoplectic attack occurring in a young person would suggest syphilis. Antisyphilitic treatment with improvement would also suggest syphilis.

Prognosis.—The prognosis in cerebral gumma is good, though relapses are frequent. The prognosis in syphilitic endarteritis for complete recovery is unfavorable.

Treatment.—Treatment should begin as early as possible, hence the necessity of an early diagnosis. Even in cases of doubt the antisyphilitic treatment should be given. Mercurial inunction should be used very freely, and potassium iodide must be given in large doses. Tonics, good nutrition, electricity, baths, and the best hygienic surroundings are necessary along with the specific treatment.

QUESTIONS.

What is the synonym of abscess of the brain?
What is the etiology of abscess of the brain?
What is the pathology of abscess of the brain?
What are the symptoms of abscess of the brain?
What is the diagnosis of abscess of the brain?
What is the prognosis of abscess of of the brain?
What is the treatment of abscess of the brain?
What varieties of cerebral tumors are met with?
What is the etiology of cerebral tumors?
What is the pathology of cerebral tumors?
What are the symptoms of cerebral tumors?
What is the diagnosis of cerebral tumors?
What is the prognosis of cerebral tumors?
What is the treatment of cerebral tumors?
What is the etiology of cerebral syphilis?

What is the pathology of cerebral syphilis?
What are the symptoms of cerebral syphilis?
What is the diagnosis of cerebral syphilis?
What is the prognosis of cerebral syphilis?
What is the treatment of cerebral syphilis?

CHAPTER V.

DISEASES OF THE CEREBRAL SUBSTANCE.

DISTURBANCES OF THE CEREBRAL CIRCULATION.

HYPERÆMIA.

Synonym.—Congestion of the brain.

Etiology.—The hyperæmia may be either active or passive.

Active hyperæmia may be caused by mental overexertion, chronic plethora, and by the constant use of alcohol, tobacco, or amyl nitrite, or from some reflex disturbance, as gastric irritation.

Passive hyperæmia may be caused by some local obstruction to the return of blood from the brain, as from pressure on the superior cava by aneurisms and tumors; from obstruction to the general circulation, as in mitral stenosis and emphysema. Prolonged mental and physical exertion with excesses and irregular living may cause it.

Pathology.—There is an engorgement of the veins of the meninges and of the brain-substance.

Symptoms.—In **active hyperæmia** the symptoms begin with a sense of warmth in the head. The face is red; the arteries in the neck are strongly pulsating. The patient complains of violent headache, ringing in the ears, is very restless, and his sleep is disturbed.

In the **passive form** the headache is not so violent. If the hyperæmia reaches a high grade, there may be stupor, drowsiness, and dulness of intellect, and finally coma resembling a slight apoplectic attack.

Prognosis is favorable when the cause may be removed.

Treatment of Acute Hyperæmia.—The patient should be

kept as quiet as possible in a dark, well-ventilated room. The head and shoulders should be raised; an ice-bag put to the head, leeches to the temples or mastoid processes, or wet cups to the neck; saline purge to relieve the constipation as well as the congestion. Give potassium bromide and aconite as sedatives.

Treatment of Passive Hyperæmia.—Try to remove the cause if possible. Pay special attention to the general condition of the patient, such as prescribing light and nutritious diet; interdict alcohol; keep the bowels regular, and correct the circulation by the use of bromides and aconite. In the apoplectic attacks venesection may be necessary.

CEREBRAL ANÆMIA.

Etiology.—Temporary or permanent diminution in the blood-supply of the brain may be caused by cardiac disease, end-arteritis, general anæmia, and mental excitement. It also exists after sudden removal of ascitic fluid from the abdominal cavity and after ligation of the common and internal carotid arteries.

Pathology.—The membranes, and, on section, both the gray and white matter are very pale. The smaller sized vessels are empty.

Symptoms.—The symptoms are known as the "fainting spell." There are dizziness, ringing in the ears, and spots before the eyes; nausea and occasionally vomiting precede the loss of consciousness, which may be absent a variable time. The face is pale, there is dilatation of the pupils, the pulse is small, and the body is covered with cold perspiration. In extreme anæmia there may be convulsions and coma. In the more chronic forms of cerebral anæmia mental effort is difficult, the patient is easily excited and irritated, is constantly drowsy, and complains of giddiness and noises in the ear, or there may be hallucinations or delirium.

Prognosis is favorable when the cause may be removed.

Treatment.—In ordinary syncope the recumbent posture alone may suffice to restore the patient to consciousness, and

should be followed by mild stimulants and rest. In other cases amyl nitrite, ammonia, and strychnine (diffusible stimulants) are necessary. Try to remove the cause: if chronic endocarditis, digitalis, strophanthus, and strychnine are indicated; if general anæmia is present, give iron, arsenic, and quinine, and manage the case accordingly in all details.

CHRONIC HYDROCEPHALUS.

Definition.—Chronic hydrocephalus is a condition in which there is a progressive accumulation of fluid within the ventricles of the brain or sometimes external to the brain and between its membranes.

Etiology.—Hydrocephalus may be primary or secondary.

Primary hydrocephalus dates from birth or develops in the first few years of life. Its cause is unknown; some authors claim that excessive overwork and worry on the part of the mother predispose the occurrence of primary hydrocephalus.

Secondary hydrocephalus follows meningeal inflammation, or the compression from tumors or some other mechanical cause producing stasis in the veins draining the ventricles. Some authors claim that syphilis is the chief etiologic factor. It may develop at any period of life. The affection seems to be hereditary in some families.

Pathology.—The head is enlarged sometimes to an enormous size, the cranial prominences bulge out, the cranial bones become thinner and flattened out, the fontanelles are widely separated, the ventricles are greatly distended with a colorless fluid of a low specific gravity, containing a trace of albumin and sometimes sugar. The ependyma is often thickened, infiltrated with leucocytes, and of granular appearance. The convolutions of the brain are flattened and the sulci more or less obliterated. Sometimes the ventricles are so distended that the effects of the great pressure cause the brain-substance to become very thin and the line of demarcation between the gray and white matter to disappear.

Symptoms.—Occasionally hydrocephalus is apparent immediately at birth and interferes with delivery of the child; but

in most cases the head begins to swell a few weeks after birth, the increase being very rapid. The enlargement is usually symmetric; the sutures are separated, and sometimes fluctuation may be detected through the fontanelles; the cranial bones are expanded and thinned until sometimes they present a parchment-like sensation to the touch. The face is small, and the head, being heavy, easily falls forward or backward. The scalp is stretched and thin, and shows very little hair; the superficial veins are engorged; the eyes protrude and are directed downward from the pressure of the fluid on the supraorbital plates. The child is very slow in intellectual development. Symptoms of motor disturbances and motor irritation arise sooner or later, but sensation nearly always remains perfect. The reflexes are exaggerated; there may be a spastic paralysis of one or more of the limbs. There is a marked general emaciation. Convulsions may occur and be repeated in many cases.

Diagnosis.—In cases not well marked, hydrocephalus must be distinguished from rickets and hypertrophy of the brain.

In **rickets** the head is square or angular and often nodular; there is no impairment of intellect; absence of motor phenomena and the presence of bony enlargements at the ends of the long bones, etc., will distinguish the two.

Hypertrophy of the brain does not develop so rapidly, the head is not so large, fluctuation is never present, the head is not so soft to the touch and compressible, and there is absence of the pathognomonic facies that is observed in hydrocephalus.

Prognosis.—The prognosis is unfavorable. Complete recovery is practically unknown. Most children gradually succumb to the affection, although a temporary or permanent arrest of symptoms is possible. Death usually results from inanition, convulsions, or some intercurrent disease.

Treatment.—The treatment of chronic hydrocephalus by internal remedies is unsatisfactory. If there is any history of congenital syphilis, potassium iodide should be given, and in other cases, if not contraindicated, it should be tried.

The surgical treatment is compression of the skull by strips of adhesive plaster and occasionally aspirating a small

amount of fluid and afterward reapplying the plaster, always carefully watching the effects of the pressure. Other surgical procedures are incision with drainage, puncture by the trocar, and lumbar puncture. Careful attention should be given toward increasing the nutrition of the patient. Best hygienic and dietetic measures, tonics, codliver oil, and massage are indicated.

QUESTIONS.

What are the disturbances of circulation in the brain-substance?
What is the synonym of cerebral hyperæmia?
What is the etiology of cerebral hyperæmia?
What is the pathology of cerebral hyperæmia?
What are the symptoms of cerebral hyperæmia?
What is the prognosis of cerebral hyperæmia?
What is the treatment of cerebral hyperæmia?
What is the etiology of cerebral anæmia?
What is the pathology of cerebral anæmia?
What are the symptoms of cerebral anæmia?
What is the prognosis of cerebral anæmia?
What is the treatment of cerebral anæmia?
What is chronic hydrocephalus?
What is the etiology of chronic hydrocephalus?
What is the pathology of chronic hydrocephalus?
What are the symptoms of chronic hydrocephalus?
What is the diagnosis of chronic hydrocephalus?
What is the prognosis of chronic hydrocephalus?
What is the treatment of chronic hydrocephalus?

CHAPTER VI.

GENERAL AND FUNCTIONAL CEREBRAL DISEASES.

EPILEPSY.

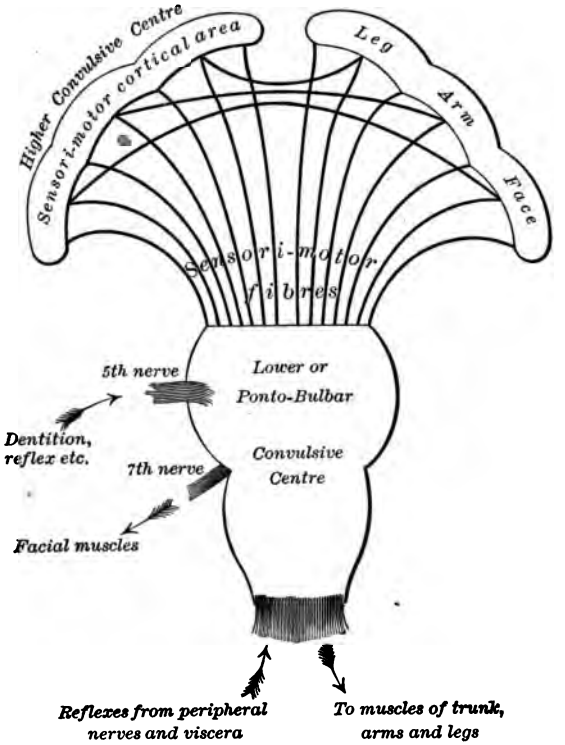
Synonyms.—Morbus sacer; Falling sickness.

Definition.—A nervous affection characterized by recurring convulsive attacks, occurring suddenly and at irregular intervals, and accompanied by partial or complete loss of consciousness.

Etiology.—**Heredity** plays an important rôle in the production of the disease. Besides epilepsy, insanity, migraine, alcoholism, consanguinity of parents, and hysteria are among the more

common ancestral taints observed. The greatest tendency to the development of the disease is shown about the time of puberty, and is rare after the twenty-fifth year. Females are

FIG. 24.



Schematic representation of the two convulsive centres, one cortical, the other pontobulbar, which may be acted upon by direct lesions or by toxic blood states; or occasionally by reflex irritations conveyed along sensory fibres from distant parts of the body.

rather more liable to be affected than males. All factors which impair the health and exhaust the nervous system are **predisposing causes.** It may follow any of the acute diseases

of childhood, injury to the head, bodily or mental excitement, anæmia, and syphilis. As **reflex causes** may be mentioned intestinal parasites, phimosis, adherent prepuce or clitoris, stenosis of the internal os, delayed menstruation, masturbation, and adenoid vegetations of the pharynx. In those subject to convulsions, overwork, gastric irritation, or excitement may precipitate an attack.

Pathology.—No decided anatomic change has as yet been found in the brain or nerves of epileptics. The following are the conclusions reached by Gowers.[1] "The muscular spasm is to be regarded as the result of the sudden overaction (discharge) of nerve-cells; the violent liberation of nerve-force and the sensations which the patient experiences before losing consciousness must be due directly or indirectly to the same cause. The disease which excites convulsions is most frequently at the cortex; and when organic disease causes convulsions that begin locally, the disease is almost invariably at the cortex. In idiopathic epilepsy the convulsions sometimes begin in this way, and this suggests very strongly that in such cases the change occurs in the cortex. Epilepsy must then be regarded as a disease of the gray matter, most frequently of the gray matter of the cortex."

Symptoms.—Two distinct types of epileptic seizures are met with: the **major attacks**, or **grand mal**, in which there are severe convulsions, with complete loss of consciousness, etc.; and **minor attacks**, or **petit mal**, in which the convulsive movements are slight and may be absent, and in which the loss of consciousness is often but momentary or practically absent. In some the attacks are diurnal, in others nocturnal, and may not be noticed for a long time.

The **characteristic paroxysm of grand mal** is usually ushered in by the prodromal "**aura**," but the onset may be sudden without premonition. The aura may be **motor**, such as a local spasm of the hand, face, or leg; it may be **sensory**, such as numbness and tingling in the face, tongue, in one finger, in the arm, in the leg, or in the stomach; an unpleasant odor, sparks or flashes of light appearing before the eyes, a mo-

[1] Diseases of the Nervous System, American Ed., 1888, p. 1098.

mentary loss of hearing, or a peculiar taste in the mouth; it may be **vasomotor**, beginning with cold and warm sensations, a flushing of the face, or profuse perspiration; or the aura may be **psychic**, consisting of mental excitement, vertigo, or confusion of ideas. The duration of the aura is variable. It is usually brief, and is followed very quickly by the seizure, but it may be more or less prolonged or, though brief itself, may precede the convulsions by many minutes.

The **convulsions** begin suddenly and at first are **tonic**. The patient falls unconscious regardless of the surroundings, and the unconsciousness may be preceded or attended by an involuntary peculiar piercing cry. The head is retracted and often strongly rotated to one side. The fingers are clenched over the thumb; the extremities are rigid. The spasm of the muscles of mastication causes the biting of the tongue. The breathing stops, the face is cyanotic, and the pupils are dilated. There may be an involuntary escape of urine and feces, which may be due to a contraction of the walls of the bladder and rectum, or to the spasm of the abdominal muscles. The violence of the muscular spasm in this stage may be very great; it has caused fracture of bones, rupture of muscles, and even dislocation of joints. The usual duration of the tonic spasm is from ten seconds to a half-minute, and is succeeded by clonic spasm. In this stage the symptoms are those of an ordinary attack of convulsions. The muscular contractions are violent and there is often frothing at the mouth. The muscles of respiration gradually relax, air enters the chest, and the cyanosis passes off.

The **stage of clonic convulsions** lasts a few minutes, and is succeeded by coma, which gives way to quiet sleep lasting sometimes for a few hours, and the patient on awakening remembering nothing or very little of anything which occurred subsequent to the beginning of the convulsion. Headache, exhaustion, a sore tongue from biting it during the convulsion, and pain in the muscles are apt to follow the attack. More rarely the convulsion is followed by a slight temporary paresis or aphasia, hysteric phenomena, vomiting, and intense hunger.

The Characteristic Seizure of Petit Mal.—In petit mal both motor spasm and unconsciousness are involved in far less degree. The attack may consist of a transient loss of consciousness alone, without motor spasm, or of motor spasm unattended by perceptible loss of consciousness. There is no fall, but there may be a slight drooping of the head, a fixed stare for a moment or two, and sometimes sudden jerkings in the limbs, and that is all. This may or may not be preceded by an aura. After the attack the patient may be dazed for a few seconds and do or say strange things, which may seem to be volitional. All sorts of odd actions may be performed, in rare instances even acts of violence may be done. Petit mal may be a forerunner of grand mal or may alternate with it.

In **Jacksonian epilepsy**, which is also known as **cortical** or **partial epilepsy**, consciousness is retained and the motor spasm may be limited to one limb or to one set of muscles, or even to a single muscle.

In cases in which the attacks follow each other with great rapidity and consciousness is not regained, the condition is termed **status epilepticus.**

Between the two extremes, grand mal and petit mal, the seizures manifest all grades of severity. Different patients may present any number or variety of epileptic seizures. The frequency of the paroxysms varies considerably; the attack may not be repeated for years, or several attacks may occur daily. A marked periodicity in their recurrence is frequently noticed. Between the attacks the epileptics seem to be quite well. After attacks have recurred during many years the intellect is apt to become permanently deranged and various cerebral disorders may develop.

Diagnosis of Grand Mal.—In most cases there is little difficulty in recognizing the major attacks when they occur by day. The biting of the tongue, blood upon the pillow, evacuation of the bladder or rectum, and the severe headache will aid in diagnosing the nocturnal attacks. **Uræmic convulsions** are recognized by the existence of greatly increased arterial tension and the condition of the urine. To distinguish grand

mal from **hysteria** is at times quite difficult, but in the latter the disease-picture simulated is nearly always overdone.

Diagnosis of Petit Mal.—The minor seizures should be distinguished from attacks of **syncope.** In the latter there is no motor spasm and no actual loss of consciousness.

Prognosis.—Death during the fit rarely occurs, but it may happen, and is generally due to some accident, particularly drowning, at the time of the seizure. Frequency of the attacks and marked mental disturbances are unfavorable indications. Epilepsy due to syphilis, if promptly treated, may be cured. A few cases of traumatic epilepsy have been cured and many have been greatly improved by a surgical operation.

Treatment.—The first indication is to **remove the cause** when one may be found. In phimosis, circumcision should be done. Adenoid growths should be removed, and likewise every other cause of reflex irritation. Special attention should be given to the digestive organs. The diet should be light and given at fixed hours. A vegetable diet is recommended by some. The patient should be placed under the best hygienic conditions. Undue mental and physical excitement should be avoided. Systematic exercise and tonic baths at proper intervals, keeping the skin active and assisting in general nutrition, are valuable. The most reliable drugs are the bromides: the potassium salt is the most effective. They should be given in large doses and continued over a long period. Bromides combined with antipyrin or phenacetin may be of great advantage. Various other drugs, as animal extracts, antitoxins, etc., have been recommended. Where the bromides fail, picrotoxin, sulphonal, trional, borax, and belladonna have sometimes proved useful. When the attack is preceded by an aura, the inhalation of amyl nitrite may cut short the seizure. Where epilepsy is dependent upon an organic or traumatic lesion of limited extent, **surgical interference** is indicated.

Treatment of the Attack.—As the seizure is short, special treatment is rarely required other than preventing the patient from injuring himself. The clothing should be loosened; a

spool or cork should be placed between the teeth to protect the tongue.

In the **status epilepticus**, chloroform and amyl nitrite inhalations, and hyoscine hydrobromate and morphine hypodermatically, are indicated.

Treatment of the Recovery from an Attack.—When possible, seclusion in quiet surroundings, a darkened room, freedom from bodily and mental irritation, together with suitable nervous sedation, is indicated. Many victims of grand mal pass at once into a deep sleep and require only to be left alone until they awake naturally.

CHOREA.

Synonyms.—St. Vitus's dance; Chorea minor.

Definition.—Chorea is a nervous affection occurring especially in children, and characterized by an irregular spasmodic contraction of a clonic kind of the voluntary muscles, ceasing during sleep, and but slightly under the control of the will.

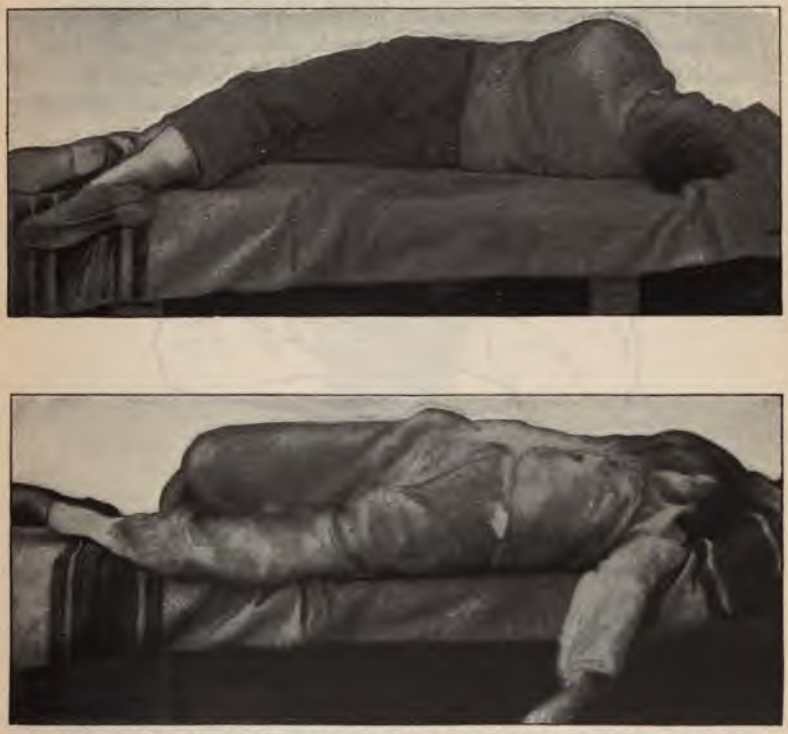
Etiology.—Chorea is most frequently seen between the ages of seven and fourteen years. It is extremely rare before the third year, although it may occur even in infancy. It is more than twice as frequent in females as in males. It is much more common in the spring months, and is more often met with in urban than in rural populations. A rheumatic diathesis, a neurotic constitution, anæmia, and infectious diseases are important etiologic factors. It sometimes develops suddenly after mental or emotional excitement, such as overwork at school, fear, grief, or religious emotion.

Among the **reflex causes** may be mentioned phimosis, intestinal parasites, delayed menstruation, adenoids of the pharynx, and enlarged tonsils. It seems to be hereditary in some families. It is sometimes induced by imitation, and the disease has been known to occur epidemically in institutions.

Pathology.—The exact pathology of chorea is at the present time not settled. The seat of the morbid process is undoubtedly the central nervous system, probably the motor areas of the cortex. In some severe cases which were fatal,

endocarditis and emboli in the minute cerebral vessels have been discovered, but their relation to chorea has not yet been determined. A specific microbic origin has been suggested, but is as yet not demonstrated.

FIG. 25.

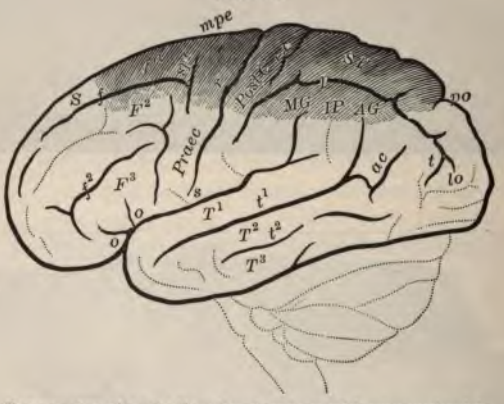


Choreic tic, showing attitudes assumed during the spasmodic movements.

Symptoms.—The onset of chorea is usually gradual. At first the child is often considered simply as unusually nervous. It is restless and awkward in its movements. At school there is noticed a difficulty in writing, drawing, etc. The

child is constantly dropping things, and has difficulty in feeding himself. It can not sit still, has a tendency to fidget, is constantly jerking its head, shuffling its feet, and there is twitching of the muscles of the eye. The child may be punished for these unfortunate conditions. **When the disease has fully developed,** the choreic movements are quite characteristic. They are irregular, jerking, spasmodic, involving the muscles of the face and head, neck, trunk, and extremities, usually more common in the face and arms, and appear in most cases only on one side of the body. The movements

FIG. 26.



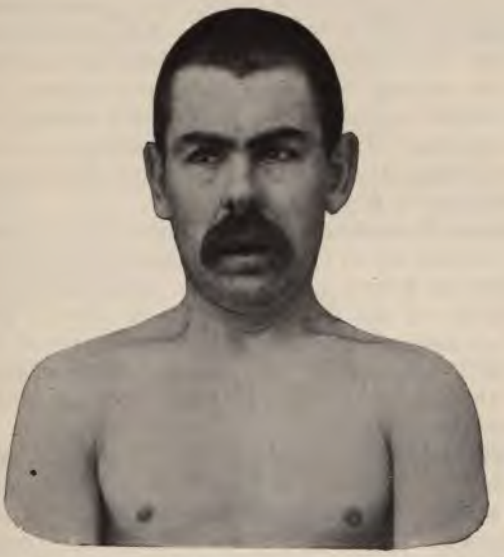
Progressive choreic tic, showing area of leptomeningitis.

vary in intensity from an occasional muscular contraction to almost constant motion. When the facial muscles are involved, they result in sudden grimaces and facial twitching; sudden closure and opening of the eyes and mouth; involvement of the arms results in twisting movements of the arms, and in attempting to grasp an object the patient may suddenly seize it and immediately drop it. The twitching may interfere with eating and dressing. When the legs suffer, the gait is usually shuffling and slow; the steps are irregular as to length and time. If the muscles of the lip and tongue are

involved, speech may be affected and articulation be indistinct; deglutition in severe cases may be interfered with and cause difficulty in swallowing; if the muscles of respiration become involved, there are uneven, irregular respiratory movements. The urine and feces may pass involuntarily.

The movements are not under the control of the patient's will, and usually are intensified by efforts to suppress them.

FIG. 27.



Physiognomy of hereditary chorea in the fifth year of the disease.

The movements are increased by excitement, embarrassment, or fatigue, but do not as a rule continue during sleep. Very often there is some muscular weakness or paresis, which may become extreme. Sensation and reflex action are normal. Some patients exhibit irritability of temper, restlessness, or peevishness. Heart-murmurs are frequent in chorea. Some of these are of anæmic origin, some possibly due to chorea of

the heart muscle itself, but a great number are due to a complicating endocarditis. The general condition of choreic patients is usually more or less impaired. They are anæmic; their appetite is poor; they sleep very badly, have frequent headaches, and are easily fatigued by slight muscular or mental exertion.

In the **violent form of chorea,** which is called "**chorea insaniens,**" the onset and progress are very rapid, the movements are violent and continuous, fever and delirium develop, and terminate not infrequently in death from general exhaustion.

Diagnosis.—There is usually little difficulty in recognizing chorea from the sudden, irregular, spasmodic contraction of the muscles coming on under the circumstances indicated. It is sometimes mistaken for **Friedreich's ataxia,** but in the latter the presence of nystagmus, the scanning speech, loss of reflexes, and the slow and peculiar incoördinate movements will differentiate it from chorea.

Prognosis.—The **ordinary form of chorea** tends to spontaneous recovery in from six to ten weeks, occasionally the symptoms may persist for five or six months. Choreiform movements of the muscles of the face, eyes, or neck may be permanent. Relapses are not infrequent. The milder forms of chorea are unattended by danger to life. **Acute violent chorea** accompanied with endocarditis may be fatal. Chorea insaniens frequently terminates fatally.

Treatment.—Should any indication of chorea appear, the child should be removed from school at once, and should never be subjected to punishment or to ridicule on account of the movements. Its attention should not be directed toward the disease. Careful search should be made for any reflex irritation. It should be placed under the most favorable hygienic conditions, and special attention given to the diet and general nutrition. Tea and coffee should be prohibited. Removal of patient from home, a trip to the country or to the seashore when possible, or almost any other change of surroundings, is frequently beneficial. A moderate amount of exercise in the open air is to be recommended. Tonics, especially iron, are indicated in most cases, as well as massage and

hydrotherapeutic measures. In all severe cases the "rest treatment" should be employed. Among the drugs that experience has shown to be most useful, arsenic holds the first place. Fowler's solution should be given in gradually increased doses, beginning at 3 drops and increased up to 10 or 12 drops thrice a day diluted in water. In cases in which there is a rheumatic history sodium salicylate is often efficient. Where arsenic fails, antipyrine and strychnine sometimes prove to be very beneficial. Other drugs that have been recommended are silver nitrate, the zinc salts, potassium iodide, hyoscyamine, quinine, and cimicifuga. In **chorea insaniens** opium, bromides, chloral, and even chloroform may be required to control the outbreaks.

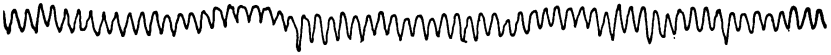
PARALYSIS AGITANS.

Synonyms.—Parkinson's disease; Shaking palsy.

Definition.—Paralysis agitans is a chronic nervous affection characterized by a continuous and involuntary tremor, muscular weakness and rigidity.

Etiology.—The affection is generally observed after the

FIG. 28.

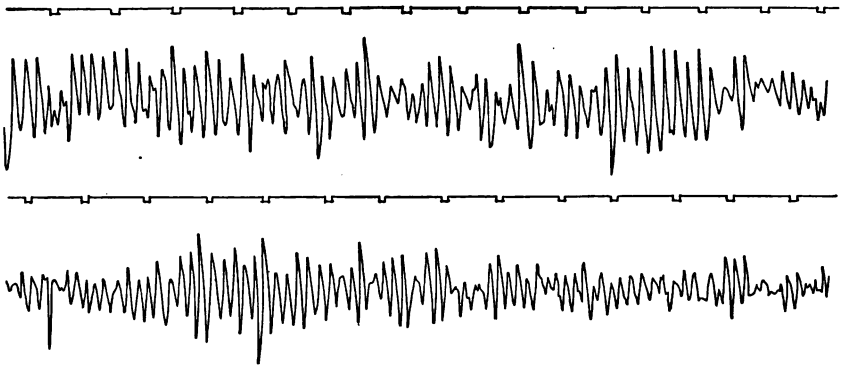


Tremor of paralysis agitans: slow revolution of drum.

fortieth year, and is more common in men than in women. Hereditary neuropathic tendencies may be traced in some instances. The exciting causes are traumatism, exposure to cold and wet, violent emotional excitement, and the specific fevers.

Pathology.—Very little of the pathology is known; post-mortem examinations have revealed no constant lesions. The lesions observed are similar to those found in cerebrospinal senility.

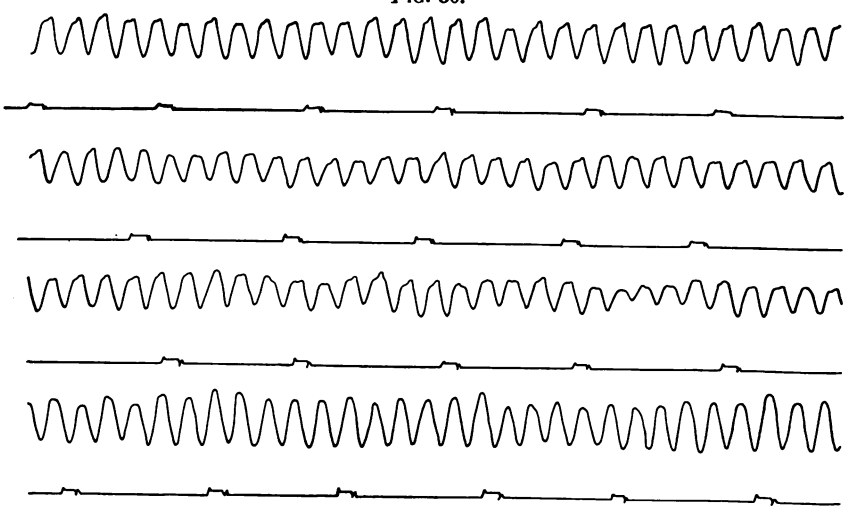
FIG. 29.



Tremor of paralysis agitans: slow revolution of drum; singular grouping of series of waves, each four seconds.

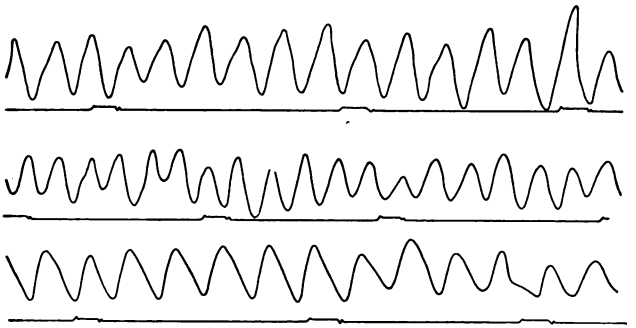
Symptoms.—Sometimes the onset is abrupt, but the disease generally develops gradually. The affection is manifested at

FIG. 30.



Tremor of paralysis agitans: more rapid revolution of drum.

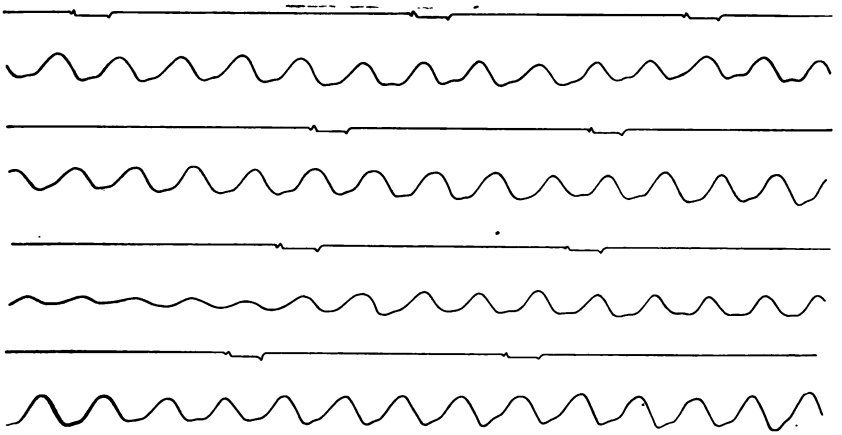
FIG. 31.



Tremor of paralysis agitans: still more rapid revolution of drum.

first by a fine tremor, beginning in the hands or feet, and gradually extending to the arms, the legs, and sometimes to

FIG. 32.

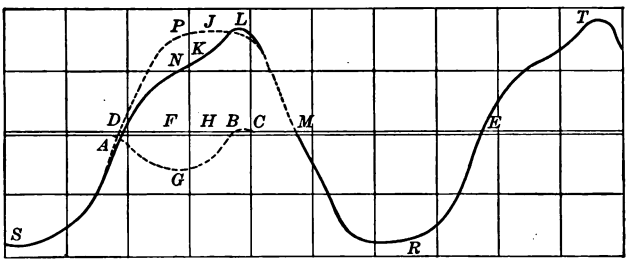


Tremor of paralysis agitans, showing dicrotism: very rapid revolution of drum.

the whole body; involvement of the head is not very frequent. The tremor consists of rapid, uniform oscillations. At first

it may be paroxysmal, but as the disease advances it is continuous. Any emotion exaggerates the movement; an attempt at a voluntary movement may check the tremor, but it returns with increased intensity. The trembling is not so violent when the body and mind are in repose, and generally ceases during sleep. The muscles become rigid and shortened; the head becomes flexed; the body is bent forward; the arms flexed; the thumbs turned into the palms and grasped by the fingers; and the legs are bent. Movements soon become impaired, and the extremities show some stiffness on motion. There is great muscular weakness, which is most marked

FIG. 33.



Geometric analysis of paralysis agitans wavelet, taken from one of the waves in Fig. 32.

where the tremor is most developed. The face is expressionless and the speech slow and measured. The gait is very peculiar, in attempting to walk the steps are short and hurried and gradually become faster and faster, the body is bent forward, the patient has to keep on running to avoid falling. This symptom is called "propulsion" or "festination." If there is a tendency to fall backward, it is called "retropulsion." The patient in bed can not voluntarily change his position. The reflexes are generally normal, but occasionally they are exaggerated. The patient may complain of a subjective feeling of excessive warmth. In other instances complaint is made of cold. The internal temperature is normal. Sometimes there is tendency to excessive perspiration. The mental condition rarely shows any change.

Diagnosis.—Well-developed cases of paralysis agitans may be easily recognized. The peculiar tremor, weakness, mask-

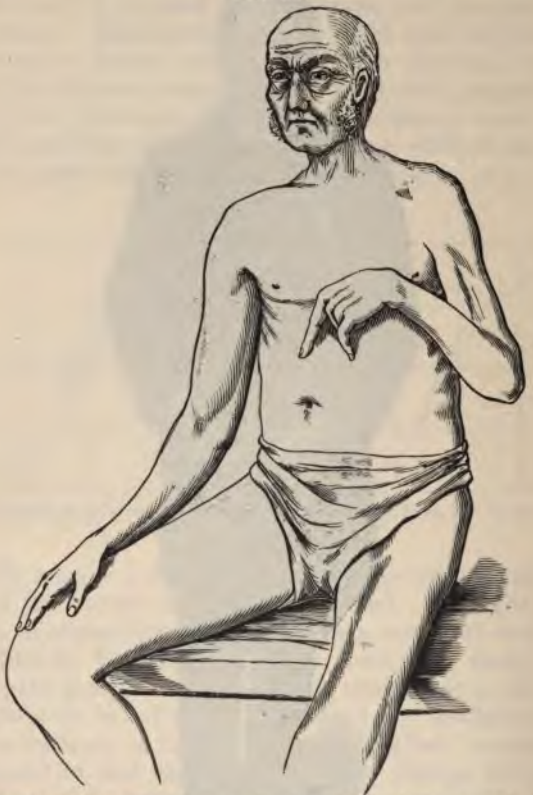
FIG. 34.

Case of paralysis agitans, showing the attitude, the position of the hands, and the facies. (Gray.)

like expression, rigidity of the muscles of the trunk and extremities, and the characteristic carriage are the important

features. The disease must be differentiated from **multiple sclerosis.** The latter develops earlier, and is characterized by the presence of nystagmus, "scanning" speech, mental im-

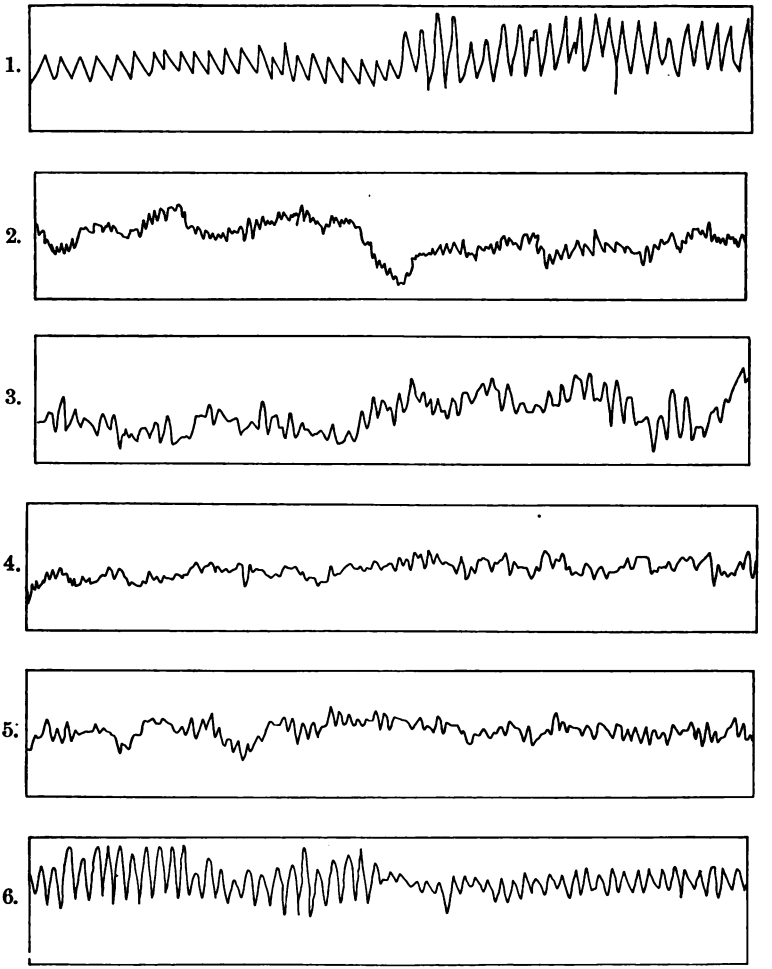
FIG. 35.



Showing wasting of left arm and leg, with contractures of elbow, wrist, and fingers, in a case of paralysis agitans.

pairment, and the absence of the peculiar attitude and gait observed in paralysis agitans. In multiple sclerosis the tremor is coarse and generally does not persist when the patient is quiet.

FIG. 36.



Comparative series of myograms of various tremors, taken with the sphygmograph, and therefore not perfectly accurate as to rate per second: 1, paralysis agitans, 4.7 per second; 2, Graves's disease, 11.7; 3, multiple sclerosis, 5.4; 4, hysteric tremor, 7.7; 5, neurasthenic tremor, 7.4; 6, delirium tremens, 5.6.

Prognosis.—The disease is incurable. It runs a very chronic course, perhaps for twenty years or more. Periods of improvement may occur, but the tendency is for the disease to grow worse gradually.

Treatment.—No means of controlling the disease are known, so the treatment is symptomatic. The diet should be regulated, and the patient should avoid mental worry or physical exercise. Frequent warm baths and gentle massage of the muscles should be tried. Electricity if given early may be of value. Nerve-stretching is recommended by some. Of the remedies used, arsenic seems to have given the best results. Ergotine, potassium bromide, opium, hyoscyamine, and curare are among the other drugs recommended.

QUESTIONS,

What are the synonyms of epilepsy?
What is epilepsy?
What is the etiology of epilepsy?
What is the pathology of epilepsy?
What are the symptoms of epilepsy?
What is the diagnosis of epilepsy?
What is the prognosis of epilepsy?
What is the treatment of epilepsy?
What is the synonym of chorea?
What is chorea?
What is the etiology of chorea?
What is the pathology of chorea?
What are the symptoms of chorea?
What is the diagnosis of chorea?
What is the prognosis of chorea?
What is the treatment of chorea?
What are the synonyms of paralysis agitans?
What is paralysis agitans?
What is the etiology of paralysis agitans?
What is the pathology of paralysis agitans?
What are the symptoms of paralysis agitans?
What is the diagnosis of paralysis agitans?
What is the prognosis of paralysis agitans?
What is the treatment of paralysis agitans?

CHAPTER VII.

GENERAL AND FUNCTIONAL CEREBRAL DISEASES (CONTINUED).

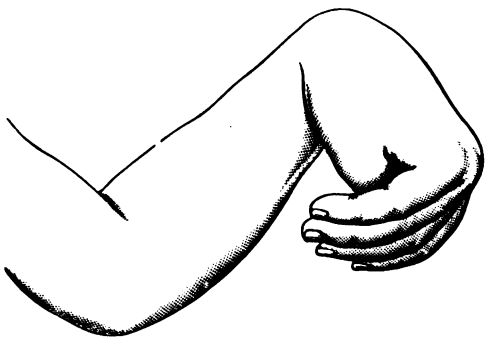
ATHETOSIS.

Definition.—Athetosis is a nervous disorder characterized by a recurring involuntary movement of a group of muscles, occurring chiefly in the hands and feet.

Etiology.—The exact causation is unknown. Falls, compression of the head during birth, alcoholism, syphilis, cerebral hemorrhage, and cerebral growths are among the etiologic factors. A few cases have been traced to heredity.

Pathology.—Some observers claim that it is due to an irrita-

FIG. 37.



The hand in tetany.

tion of the optic thalamus; others that it is due to a lesion of the motor region of the cortex.

Symptoms.—The movements may be in any set of muscles, but most commonly affect the hands, the fingers being constantly separated, extended, flexed, and approximated. The toes are also frequently involved. It may affect both sides, but in most cases it is unilateral. The movements are not always

limited to the fingers and toes, but may involve the muscles of the arm and leg and occasionally those of the face. These movements are often preceded or accompanied by other symptoms of cerebral disease, especially epileptic seizures and impairment of intellect. There are sometimes present hemianæsthesia and other perversions of sensation.

Treatment.—Athetosis is generally not influenced by treatment, though in functional cases good results have been

FIG. 38.

Showing facial spasm on the right side, the result of percussing the cheek at the point of exit of the facial nerve (Chvostek's symptom). (Thompson,)

reported from the use of arsenic and iron with potassium bromide. The galvanic current has been recommended. Hammond has obtained good results from nerve-stretching.

TETANY.

Synonyms.—Tetanilla; Intermittent tetanus.

Definition.—A nervous affection characterized by peculiar bilateral tonic spasms, which may be intermittent or continuous, and involve the extremities.

Etiology.—While tetany may occur at any age, it is most frequently observed in the young. In women it is frequently associated with pregnancy or lactation. It is frequently associated with rickets, and sometimes occurs with chronic diarrhœa and with marasmus. The acute infectious fevers, exposure to cold, and emotional excitement are sometimes the exciting cause. Tetany has been known to occur epidemically in much the same way as chorea. It may follow removal of the thyroid gland, and has been observed in gastric dilatation, particularly after the organ has been washed out.

Pathology.—Up to the present time no constant anatomic lesions have been demonstrated in tetany. It is probably a neurosis dependent upon disturbances of nutrition in the nerve-cells of the spinal cord and medulla.

Symptoms.—The spasm may occur quite suddenly, or it may be preceded by a sensation of weakness and pain all over the body. After a few minutes or hours the patient is seized with bilateral tonic spasm, usually beginning in the upper extremities, the spasm gradually becoming more severe, and finally involving the lower extremities. The fingers are bent at the metacarpophalangeal joint and the phalanges extended, pressed close together, and the thumb is contracted in the palm of the hand. The wrist is flexed, the elbows are bent, and the arms are folded over the chest. If the lower extremities are involved, the feet are extended and the first phalanges of the toes are flexed, and the second and third rows extended. The muscles of the calf are rigid and the plantar fascia tense. The tendo Achillis stands out prominently. The muscles of the face and neck are less commonly involved, but in severe cases there may be trismus, and the angles of the mouth are drawn downward.

The spasm may be excited by pressure upon the large nerve-trunks and arteries of the affected limb. This is known as "**Trousseau's symptom,**" and is characteristic of the disease. Severe pain is sometimes present during the spasm, and is increased in any attempt to overcome the spasm. There may be slight œdema of the skin of the hands and feet. Sensations and mental functions as a rule are not impaired.

The electrical reactions are generally increased, and the reflexes exaggerated. The spasms are usually paroxysmal and last for a variable time.

The **duration** of the disease is from a few days to several weeks.

Diagnosis.—**Tetanus** may be distinguished from tetany by the spasms in the former commencing in the jaw, and the etiologic factors are very different. In **hysteria** the contractures are usually unilateral and there is absence of Trousseau's sign and of muscular excitability.

The **prognosis** is usually favorable, though death may occur from the development of general convulsions or from the original disease which tetany complicates.

Treatment.—The condition with which the tetany is associated should be treated. For the relief of the spasm the hot bath is recommended; friction may also be employed. The drugs used to allay the spasm are chloral, bromides, and antipyrin. In severe cases chloroform inhalation may be given. Galvanic electricity has also been used with success. The thyroid extract may be tried. After the attack the patient's general nutrition should receive careful attention, to prevent relapse.

TETANUS.

Synonyms.—Lockjaw; Trismus.

Definition.—Tetanus is an acute infectious disease—excited by a specific organism, the *tetanus bacillus*, and characterized by violent tonic spasms of the muscles, with marked exacerbations.

Etiology.—Tetanus occurs as an idiopathic affection or follows trauma. Idiopathic cases are rare, but the affection may follow exposure to cold and wet. It is more common in the tropics than in temperate climates, and in the colored than in the white race. Males are more frequently affected than females. It has occurred in epidemic form among newborn children, especially in the West Indies, when it is known as **tetanus neonatorum.** It is very frequent in certain

localities, the soil in such places evidently being rich in tetanic bacilli.

Horses, cattle, sheep, and other animals may be attacked. In almost all cases of tetanus there is an injury, which may be of the most trifling character. It is more frequent after punctured and contused wounds, especially when occurring on the hands and feet.

The disease may follow surgical operations, extraction of teeth, vaccination, burns, frostbite, or any injury causing a solution of continuity of tissue sufficient to permit the entrance of the specific organism. In children wounds from various forms of fireworks are very apt to cause tetanus, possibly because the sand used in them is infected or because the children play in the soil afterward. Since the introduction of antiseptic surgery tetanus is less common than formerly.

The **exciting cause** is the *tetanus bacillus.* The tetanus bacillus appears under the microscope as rods of varying length; sometimes threads of considerable length are seen. When spores have formed, the bacillus has the characteristic appearance of a drumstick, enlarged at one end, in which enlargement a distinct, highly refractile sphere may be seen. It is motile, grows at the ordinary temperature, and is anaërobic. The bacilli develop at the site of the wound and do not invade the blood and organs, but produce a toxin which causes the symptoms.

Pathology.—No characteristic lesions have been found in the cord or in the brain, but the nerves in the vicinity of the wound have been noted as inflamed, red, and swelled. The condition of the wound is variable; there is apt to be a small, slightly suppurating wound, with some congestion of adjacent parts. The bacillus is present in such wounds.

Symptoms.—The **incubation period** is from ten to fifteen days. The patient complains at first of slight stiffness in the neck, or of some rigidity of the muscles of mastication, or of interference with the movements of the tongue. Sometimes chilly feelings or actual chills may precede these symptoms. The wound may become tender and painful.

Stage of Paroxysm.—Gradually a tonic spasm of the muscles of the jaw and neck develops, resulting in the typical lockjaw. The facial muscles are frequently attacked, causing distortions of facial expressions; the eyebrows may be wrinkled and the angles of the mouth drawn, producing the so-called **risus sardonicus.** Gradually the muscles of the back, abdomen, and lower extremities are involved. The muscles of the back may be affected, so that during a paroxysm the patient rests on his head and heels, a position called **opisthotonos.** The spasm of the abdominal muscles may be so intense that it causes the body to bend forward (**emprosthotonos**), and sometimes there is flexion to one side (**pleuroorthotonos**). In very violent attacks the thorax is compressed, the respirations are rapid, and spasm of the glottis may occur, causing partial asphyxia. This clonic exacerbation subsides after a few minutes or sooner, but the slightest irritation is sufficient to excite it again. In the intervals some tonic spasm of the muscles persists. During the paroxysms the patient is bathed in a profuse sweat; the pulse becomes rapid (130–140); the temperature usually remains normal until just before death, when it may reach 109° or 110° F. The paroxysms are associated with intense pain. The intellect remains clear throughout the disease. Death may occur from asphyxia, or cardiac failure, or exhaustion. The duration is from a few days to several weeks.

The **head tetanus of Rose** is generally caused by a wound on one side of the head, and the characteristic symptoms are trismus, dysphagia, and paralysis of the facial muscles on the same side as the wound.

Diagnosis.—**Strychnine-poisoning** involves the muscles of the jaw late; in the intervals between the paroxysms there is complete relaxation of the muscles, the symptoms develop rapidly, and the history of the case is different. **Tetany** affects the hands and feet especially; the nature of the spasm is different, and the history will aid in differentiating the affection from tetanus.

The **prognosis** is grave. The mortality in traumatic cases is about 80 per cent., and in idiopathic cases about 50 per cent.

The outlook is more favorable in those cases where the spasms remain localized in the jaw and neck muscles.

The **course** of the disease is slow, and there is an absence of fever.

The **local treatment** of the wound should be excision of the wound or scar, or thorough cauterizing with silver nitrate and antiseptic cleansing.

General Treatment.—The patient should be placed in a darkened, quiet room, and attended by only one person. Every possible source of irritation should be avoided. The diet should be liquid, and if the trismus is marked rectal enemata should be employed, or the patient should be fed by a gastric tube through the nose. The spasm should be controlled by chloroform. Amyl nitrite will occasionally abort a paroxysm. Morphine hypodermatically is indicated for relief of the pain. Other drugs recommended are chloral hydrate, potassium bromide, Calabar bean, curare, and belladonna. Continuous warm baths are frequently of great benefit.

The **tetanus antitoxin** should be used in all cases, and be given as early as possible. It is probably the best single remedy now at hand, and favorable results are often noted. Injection of the antitoxin into the cerebral ventricles gives the best results.

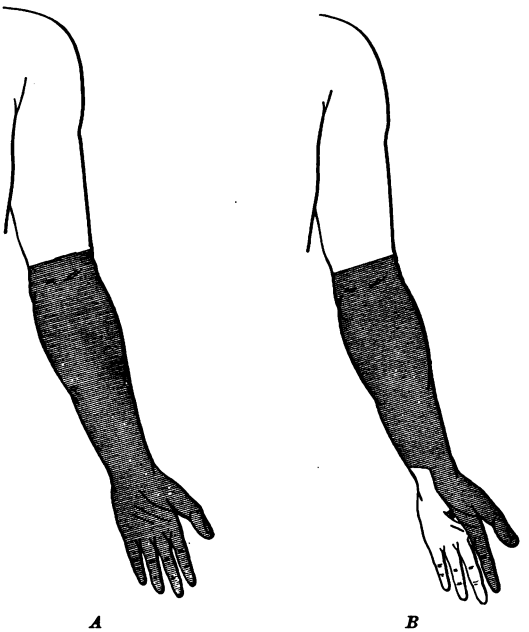
HYSTERIA.

Definition.—Hysteria is a complex disturbance of all the cerebral functions of a chronic nature, and not dependent upon any visible anatomic derangement, but intimately associated with psychic exciting causes.

Etiology.—Hysteria is most common in women, and usually appears first about the time of puberty, but the manifestations may continue until the menopause, or even until old age. Of late years it is of very frequent occurrence among men and boys. Hysteria is found among all races, but is much more prevalent in members of the Latin and Jewish races. The disease seems to be more frequent in the mild and warmer climates than in the cold. It is often hereditary in some families, and anything tending to weaken the nervous system is instrumental

in the production of hysteria. Insanity, epilepsy, alcoholism, syphilis, injuries to the head, etc., in the parents, may indirectly predispose the children to hysteria. Emotions of various kinds, fright, anxiety, grief, love affairs, and domestic worries are chief causes for the development of hysteria,

FIG. 39.

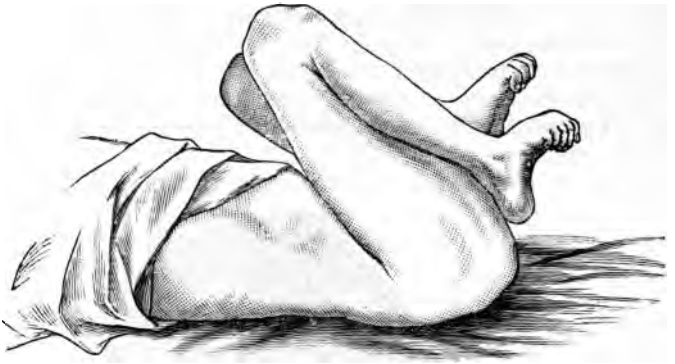


A, segmental anæsthesia of left forearm and hand; *B*, the same case four weeks later. The anæsthetic area is shaded. (Personal observation.)

especially in those of a neurotic nature. Traumatism, toxæmia, diseases of the generative organs, and organic diseases in general, and of the nervous system especially, may be the cause of hysteria.

Pathology.—Structural alterations have thus far not been

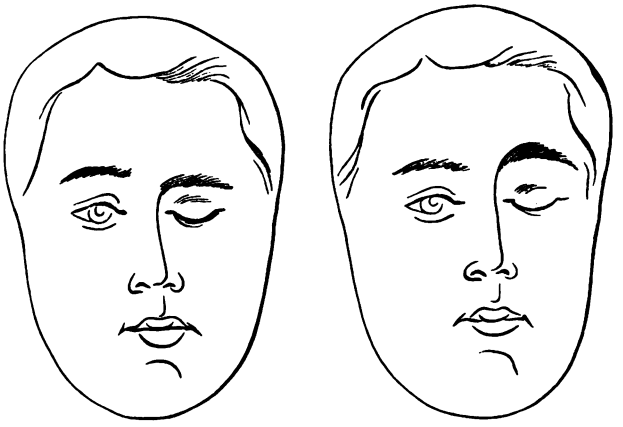
FIG. 40.



Hysteric contracture of legs.

detected in cases of hysteria. It is regarded as a functional disturbance of the nervous system.

FIG. 41.



Hysteric ptosis.

The **symptoms** of hysteria may be divided into **two classes**: (I.) the interparoxysmal and (II.) the paroxysmal.

I. The **interparoxysmal symptoms** of hysteria may be described under **four heads**: (1) psychic, (2) sensory, (3) motor, and (4) vasomotor.

1. **Psychic Interparoxysmal Symptoms.**—These are generally present to a greater or less degree in every hysteric subject. The will-power seems defective; in the worst cases

FIG. 42.

Hysteric torticollis. (Loomis.)

self-control is lost, and in all it is impaired. The patient is irritable and easily annoyed by the slightest trifle; is very excitable and easily moved to laughter or tears without any apparent cause for either. There is a great tendency to become discouraged and despondent. There is generally a great desire for sympathy. There are an increased impressionability

and suggestibility. Let an idea be suggested to the patient, especially if it relates to some physical or mental disability, and the thing suggested will likely follow. Thus one may often account for the patient feigning symptoms which really are not present. Among the more serious mental manifestations may be mentioned insanity, ecstasy, catalepsy, and trance.

2. **Sensory Paroxysmal Symptoms.**—Anæsthesia is frequently present, and the involvement of one side of the body (hemianæsthesia) is the most common form. It may involve certain parts, as one or two limbs, and the trunk escaping, or part of one limb; or various anæsthetic spots may be found in different parts of the body. In some cases tactile sensation is present, and there is a loss only of thermic or painful sensations. The skin of the affected side is frequently pale and cool, and a pin-prick may not cause bleeding. Analgesia of all tissues, skin, bone, muscle, and nerve is sometimes found. Hyperæsthesia and hyperalgesia are very commonly present in hysteria. It may be unilateral or bilateral, or hyperæsthetic spots or zones may be found in different portions of the body, and pressure upon one of these may cause a convulsive attack. A very common sensitive point is over the left ovarian region; other common points are over the breasts, lower portion of the ribs, on top of the head, and over various portions of the spine. Headache is frequently a very common and distressing symptom, and is usually situated at the vertex, but may be located in any other portion of the head. Pain in the back is also a common complaint in hysteric patients. Pains in the abdomen may be so severe as to simulate those of gastralgia and of gastric ulcer, and accompanied with such tenderness as to be mistaken for peritonitis. Neuralgic pains in various portions of the body are common. Intense pain over the heart may resemble angina pectoris. The points are frequently painful and tender. The special senses may be involved. There may be hyperæsthesia of the retinæ, contraction of the field of vision, complete blindness, disturbances of taste and smell, or complete loss of hearing. One must carefully distinguish between functional loss of power and simulation.

3. **Motor Interparoxysmal Symptoms.**—Paralysis in some form is frequently present in hysteria. It may take the form of a hemiplegia, paraplegia, or monoplegia. In hemiplegia the face is usually not involved and the leg is affected to a greater extent than the arm. Sensation is either lessened or lost on the affected side. The hysteric paraplegia is more frequent than hemiplegia. Power in the limbs is rarely absolutely lost; the legs may usually be moved, but if the patient attempts to stand the legs may give way at the knees. The reflexes may be increased; the knee-jerks may be lessened, but they are rarely ever entirely absent. True ankle-clonus is very rare, though a spurious ankle-clonus may sometimes be present. The feet are usually extended and turned inward in the position of equinovarus. The affected muscles do not waste and the electric reactions are normal. Some form of local paralysis is frequently present. Sudden aphonia, from paralysis of the vocal cords, has been noticed in many cases. There may be dysphagia from paralysis of the œsophagus, and incontinence of urine from paralysis of the bladder. The paralysis is generally paroxysmal, and is frequently associated with contractures. These contractures may attack almost any group of voluntary muscles and be of hemiplegic, paraplegic, or monoplegic type. They may come on suddenly or slowly, and may last minutes, hours, months, and some cases even many years. A condition of ataxia is sometimes seen in hysteria. The incoördination may be a marked feature. Choreoid movements of the hands, arms, or of different groups of muscles, are often seen, especially in children or young adults. A tremor is sometimes observed in hysteric patients. It may exist alone or with paralysis and contractures. It most commonly involves the hands and arms, more rarely the head and legs. The movements are small and quick.

4. **Visceral and Vasomotor Interparoxysmal Disturbances.**—The pharynx may be involved and become so irritable that deglutition is very difficult on account of the spasm of the pharyngeal muscles. The larynx may be involved and interfere with respiration. Indigestion in some form is often

present in hysteria. The stomach or intestines may become greatly distended with gas. A section of the intestine may become greatly distended and form a "phantom tumor." Constipation may be very obstinate. Persistent vomiting is sometimes present, as well as hiccoughing. Irregular and rapid heart-action is common. The slightest emotion may cause dyspnœa and pseudoänginal attacks. Rapid respiration is sometimes noticed in hysteric patients. Retention of urine is not an infrequent symptom in some female subjects. Such vasomotor disturbances as flushing of the skin, unilateral sweating, and swelling of the hands or feet, or of the joints, may occur in hysteria.

II. **Paroxysmal Symptoms.**—Convulsive seizures are common manifestations of hysteria, and frequently present a great similarity to the epileptic convulsions. **Prodromal symptoms** are frequently present in hysteria, and may begin several days before the convulsion takes place; but in the milder forms, in which the cause may be due to a temporary physical exhaustion or emotional shock, the prodromal period is of short duration. The patient may become very nervous, impatient, irritable, have fits of laughing and crying alternately, or have a feeling as of a chill rising in the throat—the *globus hystericus*—or sensation of constriction in the neck, and sometimes a painful sensation arising from the pelvic (especially from the ovarian), abdominal, or thoracic regions. These sensations resemble auræ, which are so frequently observed in epilepsy.

The **convulsions** follow these symptoms. The patient usually falls in a comfortable place; consciousness is only apparently lost, for she frequently remembers what has taken place; the tongue is rarely ever bitten. In the milder forms the movements are apt to be clonic and disorderly. In the severe forms the movements are apt to be tonic and the patient may assume the position of opisthotonos. Following the convulsive period there may be a condition of ecstasy, somnambulism, catalepsy, trance, or lethargy, or the patient may display symptoms of a delirium with the most extraordinary hallucinations. The seizures may be continued for several hours or days. Firm

pressure over the ovaries may bring on a convulsion, or if made during a convulsion it may arrest it.

The **diagnosis** of hysteria is frequently quite difficult, as it and organic disease may be associated, so that repeated, thorough, and systematic examination must be made to determine whether any organic lesion is present. The history, sex, and temperament must be carefully considered. The disease as a rule develops abruptly; it appears without obvious cause; the symptoms are generally paroxysmal; often they subside spontaneously under some emotional excitement. The health is not impaired in that proportion as usually follows the symptoms of an organic lesion.

Prognosis.—The disease is rarely dangerous to the life of the patient; yet in rare instances death has followed exhaustion induced by repeated convulsions or prolonged fasting. While hysteria usually ends in recovery, the duration of the illness is a matter of great uncertainty. Prognosis as to a speedy recovery is good in those cases in which the hysteric phenomena are connected with some obvious cause which can be removed.

Treatment.—Prophylaxis is of great importance in hysteria. It consists of proper bringing up of a child displaying hysteric tendencies. Look for some exciting cause, and if found, remove it as far as possible. The physical condition should not be neglected. Open-air life and exercise should be insisted upon as far as possible. There should be regulated and systematic habits as to eating, sleeping, exercise, and study. The mode of life should be regulated so as to insure system and order in everything. Any functional disturbance should be attended to, and tonics like iron, arsenic, strychnine, hypophosphites, and codliver oil are often indicated. Valerian, asafœtida, and sumbul sometimes give good results when mild sedatives are required.

The **moral treatment** is the most essential element, and requires great tact and skill on the part of the physician and nurse. The physician should be firm but at the same time use kindness; to inspire confidence and respect of the patient will be a great aid in bringing about a cure. Hypnotism has

been used by some with good results, but has not been tried so much in this country as in France.

The "**rest-cure**" **of S. Weir Mitchell** is of great value in hysteria, especially in well-advanced cases. It consists in isolation, rest, diet, massage, and electricity. The patient should be separated from parents and sympathetic relatives and friends; complete rest of body and mind by confinement to bed; the daily use of massage and electricity; and absolute diet of milk. The milk should be deprived of most of its cream at first; it may be diluted with soda-water or barley-water; it should be given every two hours. After a week or ten days the diet is increased, but the same amount of milk still should be given.

Treatment of Special Symptoms.—A convulsion may be arrested by the sudden use of ice to the spine or abdomen or by dashing cold water on the face and chest. Apomorphine hypodermatically will often stop a fit by the promptness of its emetic action and by the relaxation secondary to the vomiting. Inhalation of amyl nitrite will often abort a convulsion. In hysteric vomiting forced feeding by the use of the nasal tube may be of benefit. In the various forms of paralysis electricity and massage are often useful.

NEURASTHENIA.

Synonyms.—Nervous prostration; Nervous exhaustion.

Definition.—Neurasthenia is a term applied to a group of symptoms apparently resulting from functional exhaustion or debility of the tissues, especially those of the nervous system.

Etiology.—A neuropathic tendency, continued worry and overwork, sexual indiscretion, excesses, irregular living, and indiscretion in diet are general predisposing factors.

Symptoms.—The most prominent symptom of neurasthenia is fatigue. The patient complains of feeling very tired and of being unable to do mental labor; is unable to concentrate the mind on one subject for any length of time; complains of headache, vertigo, insomnia, and there are a depression of spirits, irritability of temper, and hysteric manifestations.

Sometimes there are marked spinal symptoms; there is pain along the spine, accompanied by areas of tenderness. Lumbago and various muscular pains suggesting rheumatism are frequent; there may be marked muscular weakness, great prostration after the least exertion, and various subjective phenomena, such as numbness, tingling, formication, and neuralgic pains. The circulatory symptoms observed are those of anæmia as a rule, namely, palpitation, cold extremities, and sometimes violent pulsation of the aorta, venous hums, etc. Digestion is enfeebled and delayed, and is associated with atonic constipation. Gastralgia is sometimes complained of. The urine is occasionally increased in quantity; manifestations of lithæmia are sometimes marked. Phosphaturia, oxaluria, and glycosuria are frequently noted. There are generally marked sexual symptoms; there may be impotence, seminal emissions at night and during defecation, and micturition may be frequent. The testicles or ovaries may be extremely sensitive to pressure. In females there may be amenorrhœa or dysmenorrhœa.

The **diagnosis** is rarely difficult. Before relegating a case to this class care must be taken to exclude organic disease and such general disorders as lithæmia. The disease is inseparably associated with cerebrospinal anæmia, hysteria, and hypochondriasis.

Prognosis.—When the cause may be removed and the patient controlled, the prognosis is favorable. Relapses are common.

Treatment.—Rest, mental and physical, diversions, nutritious though easily digested food, and removal of baneful influences as far as possible constitute the predominant features of treatment. Where there has been inactivity, regulated physical exercise will be of value; on the other hand, the weak and anæmic will require rest. In the latter cases, the plan of treatment introduced by S. Weir Mitchell, and known as the "**rest cure**," often gives splendid results.

The patient must be assured that he is suffering from no incurable disease. In all cases careful attention must be given to the diet, bathing, and clothing. Frequent bathing

with salt water, followed by friction of the skin, is recommended. Tobacco and alcohol must be interdicted, and tea and coffee used very sparingly. Tonics like iron, arsenic, quinine, strychnine, and phosphorus are often indicated.

APHASIA.

Synonyms.—Aphrosia; Alalia.

Definition.—Aphasia is a partial or total inability to express thoughts in words or to interpret perceptions.

Varieties.—Motor or **ataxic aphasia** and **sensory aphasia.**

The motor aphasia may be subdivided into **aphemia** and **agraphia.**

The sensory aphasia may be subdivided into **word-blindness** and **word-deafness.**

All these subdivisions may be again classified as to whether they are **partial** or **complete,** and **transient** or **permanent aphasia.**

Etiology.—The chief etiologic factors are cerebral softening, cerebral tumors, especially syphilitic lesions, cerebral hemorrhage, traumatism, and meningoëncephalitis. Aphasia may appear during typhoid fever, smallpox, and puerperal fever. Transient aphasia is sometimes observed after convulsions of epilepsy, hysteria, and migraine.

Pathology.—Motor Aphasia.—The lesion may be either cortical or subcortical, and is located in the left third frontal convolution. If only a part of this region is involved, the aphasia may be partial. In left-handed subjects the lesion is on the right side of the brain.

In **true agraphia** the lesion is in the left second frontal convolution, but it is usually associated with some form of motor aphasia.

Sensory Aphasia.—The lesion in **word-deafness** is in the posterior part of the first and second temporal convolutions.

The lesion in **word-blindness** is usually in the supramarginal and angular gyri of the left side.

Symptoms.—Motor Aphasia (Aphemia).—In this the voluntary act of giving expression to thought by the phonetic co-

ordination of the muscles of the larynx, tongue, soft palate, and lips is not performed. The patient is perfectly well able to recollect the name of an object, but is unable to pronounce it, as the power to transfer the word-image into sound is absent. The ataxia may be complete, when the patient can utter only separate sounds, or partial, when the words are only slightly mispronounced, and when some certain words can not be pronounced at all. In some cases, nouns only or verbs only are forgotten. One language may be forgotten and another remembered.

Agraphia is inability to write down the thoughts. In an uncomplicated case the patient is able to speak, hear, or read as usual; but if he tries to write, he finds that he is unable to do so, yet he is able to copy letters placed before him. It is usually associated with motor aphasia.

Amimia is an inability to perform pantomimic movements to aid in the expression of words. It is rarely or never found alone, but generally coexists with aphemia or agraphia, or both.

Sensory Aphasia.—Word-deafness is an inability to interpret *spoken* language. The sound of the word is not recognized and can not be recalled; but sounds such as that of an engine-whistle, or an alarm clock are heard and recognized.

Word-blindness is an inability to interpret *written* language. This is rarely total, a few words or letters being usually understood, and it generally coexists with word-deafness or motor aphasia.

Apraxia is an inability to recognize the use or import of an object, and may affect the senses of hearing, taste, smell, etc. The patient may take up a fork and have no idea of its use; the sound of a bell may no longer convey a meaning; or the taste of a dish.

Paraphrasia is an inability to use the right word in continued speech. The patient can interpret and use words, but is frequently misplacing them.

Prognosis depends on the cause. In young persons the prognosis is more favorable. Aphasia following cerebral softening is very unfavorable.

Treatment.—Treat the cause. If a syphilitic lesion is suspected, give potassium iodide and mercury. Injury to the skull, tumors, and other conditions causing pressure require appropriate surgical procedures. The patient should be taught to overcome the aphasic symptoms; the greatest care and patience are required.

QUESTIONS.

What is athetosis?
What is the etiology of athetosis?
What is the pathology of athetosis?
What are the symptoms of athetosis?
What is the treatment of athetosis?
What is the synonym of tetany?
What is tetany?
What is the etiology of tetany?
What are the symptoms of tetany?
What is the diagnosis of tetany?
What is the prognosis of tetany?
What is the treatment of tetany?
What is the synonym of tetanus?
What is tetanus?
What is the etiology of tetanus?
What is the pathology of tetanus?
What are the symptoms of tetanus?
What is the diagnosis of tetanu
What is the prognosis of tetanus?
What is the treatment of tetanus?
What is hysteria?
What is the etiology of hysteria?
What is the pathology of hysteria?
What are the symptoms of hysteria?
What is the diagnosis of hysteria?
What is the prognosis of hysteria?
What is the treatment of hysteria?
What is the etiology of neurasthenia?
What are the symptoms of neurasthenia?
What are the diagnosis and prognosis of neurasthenia?
What is the treatment of neurasthenia?
What are the synonyms of aphasia?
What is aphasia
What are the varieties of aphasia?
What is the etiology of aphasia?
What is the pathology of aphasia?
What are the symptoms of aphasia?
What is the prognosis of aphasia?
What is the treatment of aphasia?

CHAPTER VIII.

GENERAL AND FUNCTIONAL CEREBRAL DISEASES (CONTINUED).

MUSCULAR SPASM.

Definition.—A morbid movement of the muscles which is involuntary and due to motor irritation. The spasm may emanate from central irritation or may be due to peripheral reflex excitement.

Varieties.—Spasms in general may be **clonic (intermittent)** or **tonic (continuous)**.

In **epileptiform spasm** the convulsions are clonic and extend over the whole body.

In **clonic spasm** the movements of the muscles are either slight or very pronounced.

In **apoplexy** rhythmic spasms of certain groups of muscles are met with.

Tremor (slight motions constantly following one another) is met with in a great many nervous diseases. A constant wave-like contraction of the small fibrillar muscular fibres is observed in progressive atrophy.

Athetosis (a succession of various movements in the arms, head, but especially in the fingers and hand) is observed in some nervous affections of children or adults.

In **cataleptic rigidity** the muscles are no longer under the influence of the will, but assume any position given to them. If the tonic spasm affects the masseters, it is called **trismus**; if affecting the muscles of the back, bending the body backward, it is called **opisthotonos**; if attacking the whole body, it is **tetanus**.

Paradoxic muscular contraction is a slow tonic contraction caused by sudden approximation of the attachments of a muscle.

Classification.—Spasm of the motor branch of the trigeminus: the masseters become very hard and the jaws are firmly brought

together. This form of tonic spasm is called trismus, and may occur on one or both sides. Clonic spasm may be produced from reflex causes, and often lasts a long time. The affection should be treated by removing the cause, if it can be ascertained. Electricity is of great benefit. In tonic spasm resort must be had to artificial feeding. Narcotics may be used in severe cases.

Clonic facial spasm may be produced by peripheral or central causes. Short, rapid contractions in the muscles supplied by the facial nerves are observed either on one or both sides of the face. These come on in repeated attacks or last continuously. Voluntary muscular action is not impaired. In some cases the spasm affects the eyelids only, and is either clonic (blepharospasm) or tonic. The spasm may last but a few weeks or be present a lifetime.

Treatment is difficult. Best results are obtained from the use of electricity, applied daily for five or ten minutes. Internally bromides and arsenic are given. Application of the Paquelin cautery is sometimes of benefit.

Tonic or clonic lingual spasms may occur in hysteria or epilepsy, but rarely independently.

Spasms in the muscles of the neck may occur as a result of caries or nervous affection. The head is drawn forward, backward, or sideways according to the muscles affected. When the sternomastoid is affected and the head is drawn to one side, the condition is called **torticollis.**

Most cases become chronic, and are not easily amenable to **treatment,** which consists of electricity, the cautery, nerve-stretching, and application of narcotics. Mechanical support gives good results in some cases.

Spasms of the muscles of the shoulder and the upper extremity may occur from reflex or central causes. Usually a whole group of muscles is affected; more often the forearm and the fingers than the arm. **These spasms are treated** in the same manner as all other spasms.

Spasms of the muscles of the lower extremity occur mostly in affections of the brain or cord. Tonic spasms (cramps) come on in the muscles of the calves of the legs after fatigue.

When the reflexes are exaggerated a saltatory reflex spasm is sometimes noticed, which consists of a violent movement of the leg following every attempt to touch the floor with the foot. It is mostly seen in hysteric persons.

Spasm of muscles of respiration is always rare. If tonic, the diaphragm becomes immobile and there is a severe pain in its region. The clonic form (**singultus** or **hiccough**) is not uncommon, which is sometimes very persistent, especially when the phrenic nerve is affected. Electricity and nervines are of benefit in their **treatment.**

Vocational or Occupation Spasms.—Examples of these are furnished by **telegraphers' wrist** and **writers' cramp,** in which by overuse various degrees of painful muscular seizures occur.

WRITERS' CRAMP.

Synonyms.—Graphospasm; Scrivener's palsy.

Etiology.—Writers' cramp occurs more frequently in men than in women, and generally between the ages of twenty-five and forty. The predisposing causes are a neuropathic constitution, heredity, alcoholism, worry, and other weakening influences. The chief exerting cause is excessive writing, especially if done under a strain.

Symptoms.—The condition usually begins with a sense of fatigue, weight, or actual pain in the affected muscles. In the **spastic form** the fingers are seized with a tonic or clonic spasm whenever the pen is grasped. The **neuralgic form** is the same as the spastic, with the addition of severe pain and fatigue when writing. In the **tremulous form** the hand when put in use becomes the seat of a decided tremor. In the **paralytic form** the chief phenomena are excessive weakness and fatigue, which disappear as soon as the pen is laid aside. The patient may be emotional, nervous, and at times mentally depressed.

Diagnosis.—The history of excessive writing and the symptoms presented make the diagnosis simple.

Prognosis.—Guardedly favorable. The disease is chronic and progressive. If taken in time and if the hand is allowed

perfect rest, the condition may improve rapidly, but often there is tendency to recurrence.

Treatment.—Absolute rest is the essential element of treatment. The general condition should be improved by iron, strychnine, arsenic, and codliver oil. Massage, electricity, and passive movements give good results.

Similar cramps have also been noticed in piano- and violin-players, telegraphers, and tailors.

FACIAL HEMIATROPHY.

Synonym.—Unilateral progressive atrophy of the face.

Definition.—A disease characterized by progressive wasting of tissues on one side of the face.

Etiology.—The disease usually occurs between the ages of ten and twenty years, and is more frequent in females. Occasional causes are injuries and infectious fevers.

Pathology.—Only a few autopsies have been made. There have been observed chronic trigeminal neuritis or lesions of the Gasserian ganglion. Differences in development in the bony and other facial structures are present.

Symptoms.—The first phenomenon is often discoloration of the skin. This is soon followed by a slow wasting of all the tissues on the affected side of the face. The hair falls out in patches. The eye sinks in, the lid becomes narrow, and the pupil is dilated. Owing to the wasting of the alveolar processes the teeth become loose and finally fall out. Slight spasms of the muscles of mastication may occur. The tongue may also be affected. The appearance of the patient is very remarkable, the face looking as if made up of two halves from different persons.

Diagnosis.—The disease is recognized at a glance. The disease must be distinguished from infantile hemiplegia with atrophy, atrophy from gross nerve lesions, and congenital asymmetry.

Prognosis and Treatment.—The disease is rapidly progressive at first and then stationary, and is incurable.

EXOPHTHALMIC GOITRE.

Synonyms.—Graves' disease; Basedow's disease.

Definition.—A nervous disease characterized by exophthalmos, enlargement of the thyroid gland, and functional disturbances of the vascular system, chiefly tachycardia.

Etiology.—The disease is rare in men. It generally occurs in early adult life. It is sometimes observed in several members of the same family. A number of cases develop suddenly under emotional excitement, such as worry, fright, and depressing emotions.

Pathology.—In most cases no lesions are found on autopsy to account for the symptoms. The disease is regarded by some as a pure neurosis. Others believe that it is caused by a central lesion in the medulla oblongata. It is probably due to excessive thyroid action (**hyperthyrea**).

Four **cardinal symptoms** characterize this disease: enlargement of the thyroid gland, exophthalmos, tachycardia, and tremor.

Enlargement of the Thyroid.—One or both sides of the gland may be affected. The thyroid vessels become dilated, and a noticeable thrill may be felt, or even seen, and on auscultation various murmurs may be heard over the tumor mass.

Exophthalmos.—Synchronously as a rule with the thyroid enlargement one or both eyes become more prominent.

Graefe's sign consists in a failure of the upper lid to follow the eyeball when the latter is directed downward.

Stellwag's sign consists in widening of the palpebral angle. Vision remains normal as a rule, but the arteries of the retina throb and may be seen to pulsate.

Cardiac Phenomena.—Acceleration of the pulse and palpitation, 140 to 160 beats per minute, or even higher, are not infrequently observed. As a result of this the cardiac impulse may be felt over the entire chest, and in rare instances may be heard some distance from the patient (five feet in one case). The pulsation may be seen over the precordial region and over the vessels of the neck. The arterial pulse may be felt in the finger-tips. Sometimes a well-marked venous

pulse has been observed. Soft murmurs are not uncommon at the base of the heart.

Tremor.—There is a tremor of the hands or of the entire body. It is a fine, involuntary tremor, and appears early in the disease and increases vastly with excitement.

Miscellaneous Symptoms.—Marked changes occur in the nervous system. The mind may remain unaffected, but irritability, change of disposition, mental depression, melancholia or acute mania have been noted. As a result of impaired nutrition, anæmia, emaciation, and slight fever progressively increase. Glycosuria, albuminuria, or diabetes may be observed.

Diagnosis.—Bearing in mind the four cardinal symptoms first described, the diagnosis is usually easy.

Prognosis.—The disease generally runs a protracted course. A certain proportion of recent cases recover, but recovery in well-established cases is rare.

Treatment.—The general nutrition must be improved by rest, a liberal diet, and the use of such tonics as iron, quinine, and arsenic. The application of the mild galvanic current to the neck is often beneficial. When the palpitation is marked, no measures are so successful as rest in bed with an ice-bag or Leiter's tube applied occasionally over the heart, or over the lower part of the neck and manubrium sterni. The most reliable internal remedies are strophanthus, digitalis, belladonna, and ergot. Bromide of potassium is sometimes useful in controlling the nervous symptoms. The use of thyroid extract has not been successful. Operative treatment is frequently followed by excellent results.

PARALYSIS.

Definition.—A loss of motor power in the voluntary muscles of the body. When this loss of power is complete, it is called paralysis; when incomplete, paresis.

Path of Motor Impulses.—Disease affecting the cortical gray matter or the motor centres produces paralysis. The motor fibres take their beginning in the region of the central con-

volutions of the cerebrum and the paracentral lobule, and pass through the internal capsule, its posterior limb, the crusta, and the pons. Then after decussation in the medulla they progress through the opposite half of the lateral column and part of the anterior columns of the spinal cord to the motor ganglia of the anterior cornua. Thence they emerge as the anterior spinal roots, and pass to their peripheral endings. The injury along the track of the motor nerves may be localized in a certain spot, thus producing paralysis of a limited portion of the body (**monoplegia**). But when the injury occurs in the internal capsule, where the motor fibres are collected, the affection usually involves a half of the body (**hemiplegia**), namely, the side opposite to the injured spot in the capsule. In the medulla the fibres for both sides of the body lie together, and injury at this spot produces complete paralysis of both sides of the body (**diplegia, double or bilateral hemiplegia**). Paralysis of two corresponding parts may occur, as of the arms, and is also denominated **diplegia.** Paralysis of the two lower extremities is usually called **paraplegia.** Lesions of the cord produce muscular paralysis of the parts supplied by the respective nerves emanating from the cord below the seat of lesion. Paralysis of an individual peripheral nerve is sometimes met with.

Etiology.—It is customary to divide paralyses, **according to their cause,** into **anatomic,** with a known anatomic basis, and into **functional,** in cases in which there is no discoverable anatomic lesion. Any definite cause impairing the conducting power of the motor tract may produce paralysis, as inflammation, disturbances of circulation, new growths within the nervous tract or in the surrounding tissues, traumatism, the influence of poisons like lead and arsenic, infectious diseases due to poisoning from pathogenic products, inflammation of the nerve caused by exposure to cold. The causes of paralysis following attacks of hysteria, sudden emotion, disease of the sexual organs and of the intestines, can not as yet be explained from anatomic lesions.

Symptoms.—The patient complains of inability to perform certain motions. The affected sets of muscles after a while

may or may not show atrophic changes. W[illegible] *atrophy, the lesion is usually between the cortex a[illegible] cornua; when atrophy occurs, the lesion usually the ganglia of the anterior cornua or in the perip[illegible] tract.* In these cases with the atrophy of the mus[illegible] is also a corresponding atrophy of the nerves ("**dege[illegible] atrophy**"). The nerve-atrophy is demonstrated by c[illegible]nges in the normal electric reaction. Passive motion may be easily performed, or resistance due to rigidity or shortening (**contracture**) of the muscles ("**spastic paralysis**") may be met with.

Different Forms of Paralysis according to Situation.

a. PARALYSIS OF THE OCULAR MUSCLES.

Ocular paralysis may be **due to** affections of the peripheral or the central portion of the nerves. Direct injury, compression from tumors, thickened meninges, aneurism, exposure to cold, diabetes, acute diseases, locomotor ataxia, are among the causes producing it.

Symptoms.—The vision becomes double. If the oculomotor nerve is affected, the upper eyelid droops, the pupil is dilated, the eye can not move upward, downward, or inward. If the abducens is paralyzed, the eye does not move outward. If the trochlear nerve is paralyzed, rotation of the eyeball is impaired. With these symptoms there is often pain in the eye and in the supraörbital and frontal regions. The affection **should be treated** by means of weak electric currents, strychnine, and potassium iodide if of syphilitic origin.

b. PARALYSIS OF THE MUSCLES OF MASTICATION.

Masseteric and pterygoid paralysis from an affection of the third branch of the trigeminus is of rare occurrence.

Symptoms.—If it is on one side, there is a difficulty in chewing; if on both, chewing is impossible. The jaw hangs down and can not be moved. The cure of this affection depends on the primary cause.

13. **Radial Paralysis (Musculospiral Paralysis).**—After compression, direct injury, or exposure to cold, paralysis of the muscles supplied by the musculospiral nerve may occur. When the triceps is paralyzed from the use of a crutch, etc., there is loss of extension in the forearm. When the extensors in the forearm are paralyzed, the hand hangs down, adduction and abduction are difficult, flexion of the fingers is impaired, the thumb is flexed and adducted; flexion with the arm in position of pronation is difficult. Sensation is also impaired along the radial half of the back of the hand and the fingers. In long-standing cases atrophy becomes pronounced.

14. **Ulnar paralysis** often occurs in progressive muscular atrophy or after traumatism, or from pressure. Flexion of the hand and lateral motion are altered; flexion of the fingers is imperfect; adduction and abduction of the fingers are impossible, as well as adduction of the thumb. The proximal phalanges are extended, and the terminal are flexed, giving the hand a claw-like appearance (main en griffe). Atrophy of the interossei is marked. Sensation is lost over the portion supplied by the ulnar cutaneous branches.

15. **Median paralysis** mostly follows injury. Pronation of the forearm is lost; the terminal phalanges can not be flexed; the thumb can not be flexed or circumducted. There are often trophic disturbances and loss of cutaneous sensibility along the distribution of the nerve. The case may be mild or very marked. Most of the traumatic cases are not amenable to treatment. The primary cause should be removed if possible. A constant galvanic current, alternated with faradic applications, is of great benefit. Electricity as a means of treatment must be continued for quite a long time. Baths and local massage are great aids to the treatment.

e. PARALYSIS OF THE DIAPHRAGM.

Phrenic paralysis is but seldom met, except after an injury of the phrenic nerve or during attacks of hysteria. It may occur conjointly with other forms of paralysis in affections of the cord or cerebrum. The respiration is thoracic and very

rapid; the abdominal muscles do not act during inspiration or expiration. Bronchitis often develops, during which the act of coughing is imperfectly and painfully performed.

Prognosis.—If dependent on hysteria, recovery will take place; in other cases the outlook is doubtful.

Treatment.—Faradization of the phrenic nerve is sometimes beneficial.

f. PARALYSIS OF THE MUSCLES OF THE LOWER EXTREMITY.

1. Paralysis of the Gluteus Maximus and Minimus.—Abduction of the thigh is difficult. Walking up hill or rising from the sitting posture is difficult. Circumduction, rotation inward, and walking are impaired. The toes are turned outward.

2. Paralysis of the Anterior Crural Nerve.—Flexion of the thigh and flexion of the trunk are impaired. The leg, when flexed, can not be extended. Walking is difficult; standing is possible if the knee is extended. Rising from the kneeling position is impossible. The patella easily becomes dislocated.

3. Paralysis of the Obturator Nerve.—Adduction of thigh and rotation outward are impaired. The patient finds it difficult to cross the legs.

4. Paralysis of the Flexors of the Knee.—The knee can not be bent, and locomotion is difficult. The leg is rotated either inward or outward, and the ligaments of the knee are unduly stretched.

5. Paralysis of the Extensors of the Foot.—Extension of the ankle is impaired, walking is difficult, standing on tiptoe is impossible. The foot is everted, the ankle lowered, and talipes calcaneus results.

6. Paralysis of the Peronei.—The foot is inverted and the plantar arch is flattened.

7. Paralysis of the Flexors of the Foot.—Flexion, abduction, and adduction are impaired, and talipes varus soon develops.

The **treatment** of all these affections is the same as for paralysis of the upper extremity.

g. TOXIC PARALYSES.

Lead Paralysis.—This form of toxic paralysis occurs often in persons who have used articles prepared from lead. The lesions are a primary degenerative atrophy of the nerve, followed by degeneration of the muscle, which are due to toxic action of lead upon the nerves or the spinal cord.

Symptoms.—*The musculospiral nerve is the one usually affected.* Extension of the phalanges of the middle, ring, index, and little fingers becomes impaired. The extensors of the thumb and wrist become involved. In some cases the deltoid and the biceps are also affected. It occurs on both sides of the body, and the atrophy soon becomes marked. Sensation remains intact. On the gums above the teeth a characteristic blue "lead line" usually appears. Recovery occurs in most cases.

Treatment.—Remove the cause, administer salines, potassium iodide. Electricity is of great value.

Ether, alcohol, copper, zinc, mercury, and **arsenic** may also produce paralysis, but these forms are of comparatively rare occurrence.

General Ether Paralysis.—This variety of paralysis occurs comparatively rarely, when the ether as a toxic agent *per se* may be proved to be the basis of the lesions. Cases have been reported in which the anæsthetic itself appears to have been the sole causative factor, but they are very infrequent.

The type of symptoms is usually that of neuritis, very similar to alcoholic neuritis, and, like it, followed by paralyses in some individuals. The course of the condition is much the same as that of the alcoholic variety, and the disease slowly tends toward complete recovery.

The treatment comprises rest, nerve tonics like strychnine and arsenic, massage and electrotherapy of the affected muscles, combined with baths (hydrotherapy) for their general tonic effects upon the nervous system.

Special Ether Paralysis.—Wrist-drop occurs rather commonly after any general anæsthetization, and is due not to the

anæsthetic, but to the fact that the upper arm has been allowed to rest on the edge of the operating-table at the region of the musculospiral groove, where the musculospiral nerve passes close to the humerus and may readily be pressed upon, injured, weakened, or paralyzed.

The type of its **symptoms** is that of any other pressure-neuritis in its incipiency, establishment, and decline, and its **treatment** is, of course, analogous.

Prophylaxis is most important, and consists in protecting the upper arms from damage by pressure during operations.

Alcoholic Paralysis.—Strictly speaking, this form of toxic paralysis occurs rarely as a primary manifestation, but commonly as a secondary sign of alcoholic poisoning or as a sequel of alcoholic neuritis. Women are more frequently affected than men.

The **causes** of the paralysis are the extension of the toxic inflammation to actual degeneration of the nerves affected, due, of course, *directly* to the alcohol as it circulates in the blood, and *indirectly* to an essentially weak nervous organization, usually by hereditary taint.

Pathologically the lesions are the same as those of neuritis with degeneration.

The **symptoms** may point to a localized destruction of one nerve, to the greater damage of certain nerves when compared with others, or to a more or less generalized paralysis. The degree of the symptoms varies between those of a weakening and those of a total paralysis of the nerves.

The **course** of alcoholic paralysis is most commonly toward recovery. It is frequently, however, many months before full restitution of function is present.

The **prognosis** is favorable in the majority of cases if the patients will abstain from indulgence. As this is the exception, relapses are common with subsequent debauches.

The **treatment** involves careful management, full diet, support as a substitute for the alcohol, baths, electricity, massage, passive motion, and, as power returns, active use of the paralyzed muscles.

Copper, Zinc, Mercury, and Arsenic Paralyses.—Metal-

workers are liable to chronic poisoning, which practically always affects the nervous system as a neuritis, which in its final stages produces degeneration, then paralysis of a few or many nerves.

Their **symptoms, course, prognosis,** and **treatment** are greatly similar, and, on broad lines, those of neuritis with or without degeneration according to the severity of the condition.

The **diagnosis** of mercurial paralysis is aided by the characteristic red line of the gums, and the detection of arsenical paralysis by the puffy features and diarrhœa.

QUESTIONS.

Define muscular spasm and enumerate its varieties.

What is meant by the terms tremor, athetosis, cataleptic rigidity, and paradoxic muscular contraction? Describe each.

Name and describe the classes of spasm and outline their treatment.

What are vocational spasms?

What is writers' cramp? Give its etiology, symptoms, diagnosis, prognosis, course, and treatment.

State the chief clinical features of facial hemiatrophy.

What are the cardinal symptoms of exophthalmic goitre? Describe its other clinical characteristics.

Define paralysis, and give the general plan of its treatment.

What is the path of motor impulses?

Describe the various sites of lesions causing paralysis and their consequent symptoms.

Describe ocular paralysis in full.

What are the muscles involved in paralysis of mastication?

What symptoms point toward paralysis of mastication?

Give the clinical data of treatment of facial paralysis.

Take each muscle of the upper extremity in turn, and describe the features of its paralysis.

What is toxic paralysis and what are its features?

Describe lead paralysis fully and give its treatment.

What may be said as to general and special ether paralysis?

Describe alcoholic paralysis.

What are the paralytic features of copper, zinc, mercury, and arsenic paralyses?

What points aid in the diagnosis of mercurial and arsenical paralyses?

PART V.

PSYCHIATRY (DISEASES OF THE MIND).

CHAPTER I.

INSANITY.

GENERAL CONSIDERATIONS.

Definition.—To define insanity is almost as little possible as to describe a sane mind.

Esquirol says: "Insanity is a cerebral affection, ordinarily chronic, without fever, characterized by disorder of the sensibility, of the intelligence, and of the will."

Connolly says: "Insanity is the impairment of any one or more of the faculties of the mind accompanied with, or inducing a defect in the comparing faculty."

From the legal standpoint Blackstone says: "A lunatic, *non compos mentis*, is one who has had understanding, but by disease, grief or other accident hath lost the use of his reason." He further says: "Insanity is that mental condition characterized by a prolonged change in the usual manner of thinking, acting, and feeling—the result of disease or mental degeneration."

Insanity may also be regarded as disordered mental function. All disordered mental function is, however, not insanity; still, the physical disorder upon which the febrile or toxic delirium depends does not differ greatly from the underlying physical condition of insanity. The disorder of function in all cases is primarily due to a derangement of nutrition of the brain. This brings one to the fundamental

fact that in order to have disordered function of the brain one must have either disordered nutrition or structural alteration of this organ.

In order **to establish insanity** there must be departure from the ordinary way of thinking and acting; therefore all cases of sudden unconsciousness from injuries should be excluded, shocks, the delirium of fever, the abuse of alcohol and drugs, the delirium of the systemic diseases, the delusions of spiritualism, and belief in false religions.

The **physical basis of all mental action** is the cerebral cortex with its association fibres.

As no mental or physical manifestations can occur except through the medium of the brain, the brain is regarded as the organ of the mind, and any alteration in the structure or nutrition of the brain will affect favorably or unfavorably the functions of that organ. Upon this basis it may be assumed that without brains there can be no thought; and without healthy brain there can be no healthy thought.

The **functions of the cortex** are: To receive impressions from the sensory organs, to group these impressions into a unit, to retain this unit, to recall these impressions in the absence of sensory impulses, to group them logically in order to give expression to thought in speech and action, to feel emotional excitement and to exercise self-control over all mental organs. When the entire cortex is affected, delirium and insanity result.

The **morbid physical basis of insanity** is disordered nutrition of the brain in differing stages. It may be due to:

Anæmia.

Hyperæmia.

Inflammation of the brain or meninges.

Drugs or bacterial poisons.

Changes in the brain-structure, due to tumors, apoplexies, abscesses, embolism with consecutive softening.

Increase of connective tissue.

Structural alteration, hereditary or acquired.

Insanity should be regarded as a physical disease—a disease of the brain.

THE CLASSIFICATION OF INSANITY.

The first requisite for a logical study of insanity is one based upon known pathology and pathogeny of the disease. The time for a perfect classification of this sort has not yet arrived; all knowledge is still too vague or incomplete; but in the following an attempt has been made which may have the merits of a working scheme.

Up to within recent years classification of insanity was mainly clinical, which classification assumed that the mental symptoms are the chief symptoms to be observed. This classification took the natural history of disease into account. Accordingly the various forms of insanity were named with reference to their causation, and were divided into: traumatic, epileptic, syphilitic, alcoholic, toxic, rheumatic, choreic, gouty, phthisic, uterine, menstrual, ovarian, hysteric, masturbational, puerperal, lactational, climacteric, senile insanity, insanity of pregnancy, insanity of puberty, insanity of adolescence, as also—anæmic, diabetic, metastatic, nephritic, febrile, connubial; the insanity of cyanosis from bronchitis, cardiac disease, asthma, influenza, starvation, exophthalmic goitre, lead poisoning; the delirium of young children; the insanity following surgical operations, etc. But the most proper way of classifying insanity would be from a pathological basis, and the day will come when our knowledge of the physiology and psychology of the sound brain will be such as to enable us to make a perfect grouping of the various forms of insanity.

In the best and most modern grouping, as follows, there are seven classes or groups of mental disturbance, most of them clearly differentiated clinically, but all these **groups are based upon pathology or pathogeny** as far as is possible in the present status of knowledge of the subject.

Insanity may be due to:

1. To imperfect development of the brain, which may be hereditary, or acquired. To this group belong idiocy and imbecility.

2. To vicious or abnormal brain organization. These are

always hereditary. To this group belong paranoia, circular and recurrent insanity. Some cases of hysteria and epilepsy may also be included in this group.

3. To simple disturbance of nutrition of the brain, such as anæmia and hyperæmia. To this group belong the majority of cases of melancholia (depression) and mania (exaltation).

4. To microscopical structural alterations in the brain. General paresis, catatonia, consecutive dementia, senile dementia, and epileptic dementia are classed properly in this group.

5. To gross lesions in the brain. To this class belong: syphilitic insanity, postapoplectic insanity, insanity from tumors and abscesses, and insanity from cranial traumatisms.

6. To toxic substances circulating in the brain. In this class are included: acute confusional insanity, puerperal insanity; alcoholic, plumbic, and other chronic drug intoxications; uræmic insanity, postfebrile and most cases of postoperative insanity, and insolational insanity.

7. To developmental changes in the brain, nutritive or structural. In this class are placed: pubescent and climacteric insanity.

In order to understand the **abnormal functions of the brain** one should first be familiar with the normal brain. The brain is composed of a complex mass of tissues and fibres of extreme delicacy, which tissues and fibres, like the delicate parts of a machine, work harmoniously together to accomplish certain functions. Every organ of our body is directly connected with the brain convolutions, and their influence is mutual. Each group of cells is controlled by another group, and the cells in the cortex of the brain have the final or supreme control over all other groups in our body. The cell-groups in various parts of the brain are different in size and shape, and it has been shown that certain cell-groups are capable of doing the work of others. The brain has reflex and automatic actions. Normally the brain must act in a certain direction, but these actions of the brain are limited. Every mental action takes place directly through the efforts generated in the brain convolutions, but these manifestations

differ in different human beings, in different races, in persons of different education, in different ages, and in the two sexes, and heredity plays an important part in the brain-functions.

The chief functions of the brain are perception, judgment, volition, mental inhibitions, emotion and feeling, power of attention, representation and imagination, association of ideas, speech, and the moral faculties.

Dentition, puberty, adolescence, the climacteric, senility, menstruation, ovulation, coitus, pregnancy, childbirth, nursing, etc., bring into great activity, or may throw entirely out of action certain of the brain-functions.

From lack of use certain portions of the brain may become atrophied; on the other hand, excessive brain work causes a more pronounced development of the corresponding brain convolutions. Any disturbance in the blood-supply of the brain may cause a disturbance in the mental functions of the brain, as also a disturbance in the circulation of the membranes enveloping the brain itself. The presence of pathological formations within the brain itself, or any part of the cranial cavity, will cause a degeneration in the adjoining or surrounding brain-cells.

All varieties of mental disease are the result of excesses, defects and irregularities in the physiologic function of the brain, the result of innate morbid tendency in the cerebellum, or from excentric causes within or without the nervous system. The human brain responds to all outside impressions as well as to internal impulses. If therefore the brain is unsound the response to these impressions is imperfect or totally changed.

The **symptoms of insanity** may be divided into physical and psychical or mental.

1. The **physical symptoms** are referable to the circulatory, digestive, secretory, genitoürinary, and nervous syste The general nutrition is defective.

The **circulatory symptoms** include the following:

Anæmia is frequent, especially in states of dep mental confusion.

Depression of the circulation, weakened heart-action, and an apparent lack of vascular tonus are frequent, chiefly in melancholia, general paresis, and consecutive dementia. *Vasomotor spasm* is often present in paranoia, combined with *oppression of breathing*, and a sense of great *anxiety*.

Fever is not rare in acute states. It is most frequent in states of mental confusion and exaltation, but may also be present in depressive states. Fever is usually of grave significance and should always lead to a careful physical examination.

Among the prominent symptoms referable to the **digestive system** is anorexia, often leading to absolute refusal of food. The patient has a fear of food (*sitiophobia*). The fear of poisoning is a frequent symptom of paranoia. Delusions of obstruction or absence of abdominal viscera are often present in melancholia. Want of appetite is also seen in advanced dementia, and may be explained in such cases as the expression of the extreme indifference to all subjective sensations or objective impressions.

In maniacal states there is often an abnormal desire for food. This may alternate with absolute anorexia.

Persistent constipation is frequent in melancholia. Diarrhœa is comparatively rare. In many acute forms of mania and melancholia, and in the early stages of general paresis, the patient passes fæces into his clothing or the bed. This is not always due to loss of control of the sphincters, but sometimes to intention perverted, of course. In advanced dementia, paretic or consecutive, the loss of sphincteric control is usually paralytic.

Secretory signs are exemplified as follows:

Perspiration is usually diminished in melancholia. In mania *salivation* is often present.

Manifestations on the part of the **genitourinary system** are frequent in insanity. In maniacal conditions there is sometimes polyuria. *Incontinence* is frequent in acute mania and in dementia. Involuntary passage of urine often occurs during epileptic attacks. The urine may be *retained* owing to indifference, or its retention may be due to a delusion. *Ex-*

aggerated sexual desire is frequent in mania and the early stages of general paresis, leading to venereal excesses or to masturbation. The most shameless acts of exposure and solicitation are seen in females, although masturbation is probably more frequent in males. In depressive states, and in the advanced stages of general paresis sexual desire and power are diminished. Desire sometimes persists when potency is absent.

In acute psychoses *menstruation* is nearly always arrested. It is said that one of the earliest signs of improvement in acute insanity in women is a return of the menstrual flow, provided it was absent during the acute stages of the attack.

Among the **nervous phenomena** of insanity the most frequent is *insomnia.* Insomnia is sometimes very persistent in mania and general paresis, and is an occasional symptom of general paresis. Occasionally in general paresis there is a great *tendency to sleep.*

Headache is a symptom in general paresis, cerebral syphilis, and in melancholia. In the latter, occipital cephalalgia is said to be diagnostic. Headache, more or less intense, also attends most cases of mental disturbance depending upon gross lesions in the brain.

Convulsions are present in epilepsy, uræmic insanity, general paresis, and syphilitic insanity. The convulsions in general paresis and syphilitic insanity are not typical epileptic seizures, but of the character described as epileptiform. They also occur at times in alcoholic insanity. The epileptiform attacks of general paresis are usually followed by a comatose or paralytic state lasting several hours or days. These apoplectiform seizures also follow true epileptic convulsions at times.

Irregular pupils are frequent in general paresis.

Tremor is present in alcoholic insanity and in certain forms of mental disturbance complicating cerebrospinal diseases. The fibrillary tremor of the tongue and facial muscles in general paresis is diagnostic in many cases.

The *tendon-reflexes* are usually dimin al

paresis, alcoholic insanity, and peripheral neuritis. In some forms of melancholia the knee-jerk is increased.

The *speech* is early affected in general paresis, and the scanning speech of the paretic is characteristic. In dementia the speech is often indistinct or slurring.

Trophic disturbances may also be looked upon as physical symptoms of insanity. Thus, the peculiar deformity of the ear termed "othæmatoma," or "the insane ear," is almost limited to insane persons. Bedsores develop with great rapidity in the insane, especially general paretics and epileptics. A peculiar fragility of the long bones has also been noted.

2. Psychical Symptoms.—In maniacal states, paranoia, and the early stages of general paresis, the emotional instability is much heightened. The patient is easily "upset"; slight irritants may cause violent outbreaks of anger or rage with destructive attacks. In melancholia the emotional instability tends to react to painful impressions. The patient is easily moved to tears, or is subject to morbid anxiety, sorrow, or fears, which are so often present in neurasthenic states and depressive forms of insanity. The chief psychical symptoms of insanity are sensory and intellectual disturbances. The former are termed hallucinations and illusions, and the latter delusions and impulses.

Hallucination.—Hallucination is an act of perception when no object is near, and is therefore without foundation. It is in a sense a symptom of insanity as well as of delusion of the person who hears voices of people, and sees objects that are not present. He sees faces and forms of relatives long deceased, and a patient with hallucinations usually stands in an abstracted manner, or seems absorbed in deep thought, or gesticulates actively in a conversation with unseen persons. These hallucinations may be so annoying that the patient's sleep and rest are destroyed. Patients complain of seeing wicked and obscene things, that they are persecuted, that they are told to do undesirable things; they claim communication with spirits, and insist on having the ability to receive messages through the ether from all parts of the world. The various senses of taste, smell, and feeling may also be perverted.

Food does not taste naturally, or the person claims that it is poisoned, and therefore refuses to eat. Bad odors are fancied by the patient, and perversions of feeling occur, causing an uncomfortable sensation of cold, heat, or itching. Gradually the peace of mind is destroyed and criminal acts, such as suicide, homicide, and assaults, are common where persistent hallucinations are present. Most persons affected with hallucinations present an abstracted appearance, as if their attention were fixed by some imaginary object. The eye is moved about in a restless manner; their conversation is disconnected and incoherent.

An hallucination is, therefore, a false sense-perception having no objective basis. There may be hallucinations of the special senses: hearing, vision, smell, taste, or of common sensation. Auditory and visual hallucinations are especially frequent, and are often symptoms of dangerous forms of insanity.

In **hallucinations of hearing** sometimes the voice is an internal one and commands the patient to kill his persecutor or destroy the latter's property. Hallucinations of hearing are especially frequent in paranoia.

Visual hallucinations are present in paranoia, mania, and epilepsy. One of the most dangerous visual hallucinations seems to be that of "seeing red." The suggestion of blood often leads to homicide.

Hallucinations of taste are found in paranoia and melancholia. In the former the patient "tastes poison" in the food and hence refuses to eat, unless he can get food secretly. The hallucinations of taste of the melancholic are, perhaps, sometimes exaggerated perversions of taste due to digestive disturbances. The same may be said of the hallucinations of smell.

Hallucinations of smell are not rare in paranoia, climacteric insanity, melancholia, and especially in mental disturbances due to ovarian and uterine disease.

The hallucinations of the various special senses are often associated.

Hallucinations of common sensation often give rise to complaints of vermin crawling upon or burrowing in the skin.

Illusion.—An illusion is a sense-perception having an objective basis, but falsely translated to the consciousness. It is a faulty conception of an actual sense-impression.

Illusions may be called false beliefs or the distortion of actual objects by the senses. The identity of objects and of individuals is mistaken, and living and moving objects appear to the patient unreal.

Delusion.—Delusions are false conceptions and judgments without sufficient logical foundation and accepted as true. Wood's definition of a delusion is: "A faulty belief concerning a subject capable of physical demonstration out of which the person can not be reasoned by adequate methods for the time being." A faulty belief may be a delusion in one person and not in another. It is largely a matter of education, or of environment. The contrary belief is not a delusion, but simply ignorance.

A distinction is made between insane and sane hallucinations and delusions. The former are said to dominate the life and acts of the subjects, while in the life and conduct of the latter the hallucinations and delusions are merely incidental.

The insane usually act from the same motives as those that govern the sane, but they are impelled to action by erroneous beliefs and ideas. No argument or persuasion can correct these false beliefs, and in this the insane differ from the sane as the erroneous opinion of the sane may be easily corrected by experience, knowledge, or argument. The mental life of a child before its judging power is developed is one series of delusions; the superstitions of the ignorant are delusions, the result of want of judging power. Dreaming and nightmare are the nearest physiologic counterparts of insane delusions, and exhaustion or want of sleep will produce a condition closely allied to monomania. The education, age, class, and race determine to a great degree whether any given false belief is an insane delusion or not.

Varieties.—Delusions may be expansive (delusions of grandeur), depressive (delusions of debasement), delusions of persecution, and religious and sexual delusions. The delusions of grandeur and of debasement are the fundamental

varieties. Persecutory, religious, and sexual delusions are based upon some delusion of exalted or debased personality. In all phases of delusive belief the grandiose character is maintained, and the idea of persecution is merely a further development thereof.

Delusions of grandeur are present in general paresis, in which disease they have long been regarded as characteristic. They are also an essential element in paranoia, in which persecutory delusions are an outgrowth of them. In melancholia, delusions of debasement are often characteristic. In mania delusions of grandeur are often transitory and varying; in general paresis they are extremely extravagant, and in paranoia they are fixed and in a sense logical.

Delusions of debasement or of unworthiness are common in melancholia; they are rare in paranoia. In this form of insanity they are closely connected with hallucinations of hearing, smell, and taste. Persecutory delusions are extremely dangerous symptoms. Under the influence of such delusions most of the acts of violence of the insane are committed.

Religious delusions are found in paranoia, epilepsy, and melancholia. In paranoia and epilepsy they are nearly always of an expansive character. The religious delusions of melancholia are usually permeated with a profound sense of unworthiness of the subject, while the paranoiac is saturated, so to speak, with the sense of his own importance and power, and is always convinced that he is entitled to more honor than the world renders him; the melancholic, on the other hand, constantly and loudly protests his utter unworthiness, his sinfulness, the impossibility of ever regaining the lost grace of God.

Sexual delusions of an expansive character are present in the early stages of general paresis, in mania, and in paranoia. In the latter they are combined usually with persecutory delusions.

There are **mixed types of delusions**, such as *megalomania*, or delusions of grandeur, combined with those of suspicion and persecution: also the monomania of unseen agencies when patients believe that they are electrified or mesmerized, that

noxious gases are blown into their bedrooms, that people speak to them and call them bad names, that spirits haunt them, that persons come to them at night, that their thoughts are read. Some patients have pleasant delusions, especially those of a sexual character.

Morbid Impulses—Impulsive Acts.—When an epileptic during an hallucinatory aura attacks another person, a paranoiac under the influence of his persecutory delusions commits murder, or a sexual pervert cohabits with animals or with dead bodies, the acts are said to be *impulsive*, and committed in obedience to an imperative impluse, conception, or idea. Suicidal and homicidal mania, dipsomania, pyromania, kleptomania, erotomania, onomatomania, are merely coercive impulses, often irresistible.

General Diagnosis of Insanity.—The differentiation of the individual forms of insanity is difficult, but it is still more difficult at times to say with positiveness that a person under examination is sane or insane. This is largely due to the fact that there is no absolute standard of sanity. The general diagnosis of insanity must take into account not only the subject's conduct at the time being, but also his previous history and his environment.

General Prognosis of Insanity.—Insanity is curable in a considerable proportion of cases. If appropriate treatment is promptly instituted, the percentage of recovery should be about 40 per cent. If certain groups that are incurable, such as imbecility, paranoia, general paresis, and epileptic and other secondary dementias are excluded, the proportion of recoveries should be much larger. We may expect recovery in 75 per cent. of the psychoses due to nutritive disturbances or toxic conditions.

General Principles of Treatment of Insanity.—Inasmuch as insanity is here considered as a purely physical disease, it is evident that purely psychical remedies occupy a very subordinate part in the treatment. They are limited to what may be called, in a general way, the management, or handling of the patient. Agreeable surroundings and the keeping at a distance of sources of irritation may also be classed with the

psychical remedies. Total isolation is not to be recommended, especially in hallucinatory and delusional forms.

Regarding the question of **institutional or home-treatment,** the decision should in all cases be in favor of the former.

The **general physical treatment** consists in good food, rest in bed in acute cases, and out-door life after the acute stage is over and the danger of exhaustion has passed.

In the majority of cases restorative tonics are necessary.

Insomnia may generally be combated by baths, out-door life, attention to hours of feeding, proper bed-clothing, and by occasional use of hypnotics, especially if several different drugs are employed alternately.

In the comparison of narcotics and hypnotics they are ranged in the following scale: morphine, chloral, amylene hydrate, paraldehyde, and sulphonal. If arranged, however, in order of hypnotic power, and at the same time their innocuousness, they would stand in the following order: chloral, sulphonal, amylene hydrate, paraldehyde, lactophen, and morphine.

The following mixture is recommended:

℞—Potassium bromide,	2 drachms;
Chloral hydrate,	½ drachm;
Syrup of morphine (French codex, $\frac{1}{16}$ grain to the ounce),	1 ounce;
Distilled water,	3½ ounces.
S. ʒij–iv as required.	M. Luys ('95).

The hydrochlorate of morphine is of great value in the treatment of mental and nervous disorders. Excellent results may be obtained with the wet pack.

Lactophen is also given for insomnia.

Opium is rarely necessary in insanity in children; a wet pack is preferable. The administration of bromide of sodium acts as a calming agent. Open air exercise is to be prescribed in all cases. The most important feature of the treatment is the separation of the child from his friends.

The prevention of insanity in [illegible] very essential matter.

When food is refused on account of gastric derangement, lavage of the stomach and careful, systematic feeding will prove useful. The food must be varied and carefully selected with reference to the digestive powers of the patient.

Care must be taken to vary the food even when it is being given through the gastric tube.

The subcutaneous infusion of an albumin-salt solution is extremely valuable in cases of sitiophobia. The proceeding is not very painful, and leaves no bad local after-effects.

Among the means of treatment employed in acute cases of insanity, none surpasses, in effect, rest in bed. The suggestive influence of other persons in bed and apparently sick has a favorable effect, and the patient soon yields to the suggestions of physicians and nurses and regards himself as sick and in need of treatment.

Mechanical restraint and seclusion in a dark or barred room are not necessary in the treatment of insanity in any of its forms, and should not be employed except in rare instances under special indications.

QUESTIONS.

How would you define insanity?
What are the legal definitions of Blackstone of insanity?
What is the physical basis of mental action?
What are the functions of the cortex?
Describe the usual morbid bases of insanity.
What elements should be considered in order to establish insanity?
What may be said as to the difficulties of classifying insanity?
Give the best two classifications of insanity as at present accepted.
What are the normal functions of the brain which are commonly disturbed in insanity?
Describe the abnormal functions of the brain as seen in insanity.
What are the symptoms of insanity referable to each of the various systems of the body?
What are hallucinations?
Describe the varieties of hallucination.
What are illusions?
What are delusions?
Name and describe the varieties of delusion.
What are morbid impulses?
What do you say as to the general diagnosis of insanity?
Is the prognosis of insanity favorable or grave?
Describe the general principles of treatment of insanity.

CHAPTER II.

INSANITY DUE TO IMPERFECT DEVELOPMENT OF THE BRAIN.

IDIOCY AND IMBECILITY.

Idiocy and Imbecility.—These two conditions of defective mental function, are merely different in degree, both being *dependent upon* defective or arrested cerebral development. This defective development may be hereditary, congenital, or acquired; that is, it may occur in intraüterine life, during the parturient process, or after birth. Idiots and imbeciles are usually termed "feeble-minded."

A person born without mental faculties or capability is an idiot. An idiot does not become insane because the mental faculties are not developed to a sufficient degree to pass from the state of sanity into the state of disorder or disease. An idiot remains an infant throughout his whole life. Imbecility in contradistinction from idiocy is caused by partial or arrested development of the brain centres, which arrested development usually appears before the age of puberty. A child may appear perfectly normal, mentally and physically, until he reaches that age when the brain is called upon to perform a more complex work, when the child shows a lack of power to receive new impressions, displays ungovernable temper and cruelty to animals, loses all affection for those dear and near to him, has no business capacity, and may show criminal tendencies.

Idiocy and imbecility can not properly be classed with diseases of the mind. They are characteristics of certain individuals with arrested brain-development, and may be looked upon as eccentricities of certain individuals, which may not in themselves necessarily constitute a state of insanity in the full meaning of the term as at present accepted.

The proportion of feeble-minded is about 1 in 500 of population. Males outnumber females 2 to 1.

Etiology.—Idiocy and imbecility are hereditary in about half of all cases. The principal conditions in the ancestry supposed to influence the heredity are insanity, nervous diseases, intemperance, consanguinity, and tuberculosis. Intemperance in the parents is a factor in only about 10 per cent.

A considerable proportion of cases of feeble-mindedness are due to traumatisms during the process of birth. Prolonged labor, subjecting the brain to undue compression, direct traumatisms from the use of instruments or improper

FIG. 43.

Growth of beard in a female: stigma of degeneration (Incurables' Hospital).

methods of delivery; convulsions in the mother with consequent poisoning of the fœtal blood by carbon dioxide or by anæsthetics used to relieve the maternal convulsions; or premature birth may produce such a disturbance of nutrition in the brain as to arrest or retard its development.

Acquired idiocy, beginning in infancy or childhood, is due to the toxic influence of infectious diseases, to injuries, rachitis, meningeal inflammation, fright, convulsions, and improper training.

Symptoms.—The most notable is microcephaly, or abnormal smallness of the cranium. This may be due either to imperfect growth of the brain from intrinsic causes, or to premature closure and ossification of the cranial sutures.

In contrast to microcephaly, many cases of idiocy show a

larger skull than normal (macrocephaly). They are usually hydrocephalic.

Irregularity or asymmetry of the skull and brain are also present at times. Defective development of the remainder of the body is frequent.

FIG. 44.

Hydrocephalic imbecile (Incurables' Hospital).

Pareses and paralyses are among the physical symptoms often noted. Epilepsy and other forms of convulsions are frequent complications. There may be various tics, athetosis, and atrophy of paralyzed limbs. Strabismus is common.

Psychical Symptoms.—Defective intelligence is the most marked characteristic of the idiot. There may be cunning,

a retentive memory, acuteness of the special senses, and even the mathematical faculty may be highly developed in some peculiar directions, but judgment and self-control are lacking. There is nearly always defective articulation. The

FIG. 45.

Microcephalic idiot (Incurables' Hospital).

expression is generally placid and good-natured. The patient seems often to feel the necessity of guidance but at other times his expression may become brutified. Sexual instinct is often active. Masturbation is frequent, and its constant practice still further brutifies the defective subject. The uncontrolled sexual desire may also lead to offences against morality in both sexes. Sexual perversion is not infrequent. There is

often a perversity of character. The patient will strike without provocation, spit at those who endeavor to correct him, and he seems to have a special delight in soiling his clothing with excreta. The so-called "moral idiot" belongs

FIG. 46.

Diplegic idiot (Incurables' Hospital).

to the same class with the other idiots. The chief essential feature of his malady is weak-mindedness.

Diagnosis.—The symptoms in chronic dementia are progressive; the symptoms of the idiot remain unchanged.

Prognosis.—The prognosis of idiocy and imbecility is unfavorable. There is at present no means known to medical

science and art by which a brain defective in structure or organization may be made perfect.

Treatment.—The treatment should be mainly prophylactic. If anatomic defects are at the basis of the feeble-mindedness, no method of treatment known offers any chance of improvement.

The main reliance must be placed upon good pedagogic methods.

QUESTIONS.

Define idiocy and imbecility.
What is the etiology of idiocy?
What are the symptoms?
What are the physical and psychical signs of idiocy?
What is the diagnosis from chronic dementia?
What is the prognosis of idiocy?
What is the treatment of idiocy?

CHAPTER III.

INSANITY DUE TO VICIOUS OR ABNORMAL BRAIN-ORGANIZATION.

PARANOIA.

Synonyms.—Primäre Verrücktheit; Chronic delusional insanity; Monomania.

Definition.—A chronic, inherited, incurable form of insanity, generally progressive, characterized principally by primary fixed systematic hallucinations and persistent delusions, and rarely terminating in dementia, which dominate but do not impair the intellectual processes.

Etiology.—Persons burdened with an inherited neuropathic tendency usually show psychical evidences of it in early life. After puberty the eccentricities of behavior become more marked, and the patient can not live in peace and amity with any one for a long time; he develops hal-

lucinations and delusions. The hallucinations and delusions dominate the thought and conduct of the subject. From the twentieth to the fortieth years the eccentricities, hallucinations, and delusions either gradually or suddenly become more marked and become systematized.

Symptoms.—Hallucinations.—Those of hearing are most frequent and annoying. Rarely the character of the hallucinations is pleasant and agreeable; much more frequently they are irritating.

Hallucinations of vision are often of a pleasurable character. On the other hand, the visions may be disturbing or terrifying and produce delusions of suspicion or persecution in the ordinary course of the disease.

Delusions.—Delusions usually follow hallucinations. The characteristic delusions are those of persecutions, combined with delusions of grandeur. There are also delusions of personality, where the subject fancies himself another person. The delusions rather frequently are those of electrical and hypnotic influence and of thought-reading. He usually gives his complaint very extensively and often connectedly in writing. The electrical or hypnotic apparatus is also used, depriving the patient of sexual power, or compelling him to masturbation, which he regards as an attack upon his self-respect. If one does not believe his words, it is easy to prove it absolutely by a galvanometer which if attached to his head will show the presence of an electric current.

Under the influence of delusions of persecutions the patients themselves become persecutors.

The delusions of grandeur may be present with or without hallucinations. These persons are also dangerous, because they sometimes seek to obtain by force the honors of which the world has robbed them.

Paranoia may be of hypochondriacal, religious, erotic or of other variety, but no arbitrary classification may be accepted.

Course.—Paranoia does not have a perceptibly acute stage, and seems chronic from its incipiency, giving the

history of degeneration of the mental and moral faculties with a slow almost imperceptible change in the character of the individual, his affections and passions, causing the development of distinct illusions, influencing the life of the patient. It is a strongly hereditary insanity. The change from the normal mental stage of the individual is usually in the direction of elevated ideas, often accompanied by symptoms of morbid suspicion and sensitiveness. In one word the mental reaction seems to be abnormal and the inhibitory power of reason is absent so that the patient is unable to correct mistaken conclusions. The patient loses his hereditary social instincts and his hereditary morality becomes perverted, causing a perversion of all instincts, appetites, and desires. His affection changes and is disruptive in its effect, causing the patient to turn from his family and friends and acquire unnatural affection for strangers or those unequal in a social scale. Crimes are often committed under the influence of morbid delusions, and gradual mental enfeeblement ensues.

The delusions of the paranoiac are systematized and occupy his mind for a long time to the exclusion of all other thoughts, hence the name *monomania*, but as human impulses are varied, some writers have given a name for each, as homicidal monomania, suicidal monomania, kleptomania, dipsomania, etc.

The majority of paranoiacs are orderly, peaceful and contented with quiet surroundings; the fixed ideas are replaced by general delusions, and finally mental deterioration sets in.

Diagnosis.—The history of a neuropathic ancestry, the slow development, the persistent character of hallucinations and delusions, with the comparatively slight degree and late appearance of dementia differentiate paranoia from other forms of insanity.

Prognosis.—Paranoia is incurable. Remissions, and prolonged intermissions for a year or more may occur.

The duration of life is not shortened by paranoia.

Treatment.—The paranoiac is always a dangerous char-

acter, and hence requires to be kept under observation. In all cases of paranoia the patient should be placed under strict control. There is no other safe treatment.

QUESTIONS.

What is paranoia?
Give its etiology.
What are the symptoms of paranoia?
What are the diagnosis and prognosis?
What is the treatment?

CHAPTER IV.

RECURRENT FORMS OF INSANITY.

Varieties.—Recurrent or periodic insanity appears as states of exaltation (**mania**), depression (**melancholia**), or an alternation of the two (**circular** or **cyclic insanity**) with intervals of apparent lucidity. **Periodic dipsomania** is one form of recurrent insanity. The tendency to recur persists throughout life, and dementia is rare.

RECURRENT MANIA.

Symptoms.—The essential feature of recurrent mania is the occurrence of exaltation of feelings without confusion of ideas. This state may last a month or longer in the exalted stage; the patient gradually, sometimes suddenly, returns to his normal mental condition, but the victim of periodic insanity exhibits even in the intervals evidences of some involvement of the intellectual functions.

Recurrent insanity (**folie circulaire**, as the French term it) is a recurrent psychosis of variable type. In some patients the period of exaltation is long and the depression and insanity short; in others this is reversed, but in typical cases the periods are each about of the same length. Exaltation, which usually is a pure brain-exaltation, is often but hyperæsthesia

and exaltation of many of the nerve functions. The depression is often accompanied by apathy and torpor, and we rarely notice suicidal impulses. The period of sanity is usually a stupid, inactive sanity. When a patient of this class, who otherwise is quiet and regular in his habits, suddenly becomes talkative, anxious to attract attention, makes unusual speeches, and so on, we may feel sure he is on the verge of another attack. The majority of cases show a neurotic tendency, and are the result of mental degeneration, intemperance, or vices of ancestors. Puberty, the menopause, marital troubles, sudden joys or sad experiences may act as predisposing causes. The recovery from each attack is usually sudden, but as the attacks multiply recovery takes place much more slowly.

During the attack there is usually some loss of weight. The first attack most frequently occurs at puberty. In women succeeding attacks often coincide with menstruation.

Prognosis.—Permanent restoration of normal mental function does not occur. The intervals between attacks may be weeks, months, or years. Dementia is rarely a sequel.

Treatment.—Chloral and bromide of potassium may be given to depress the circulation and cerebral exaltation. Bed-rest, baths, and good feeding are essentials in the treatment and equally as important as medicinal agents.

RECURRENT MELANCHOLIA.

The **symptoms** are usually those of simple melancholia without delusions; the attacks come on rapidly and disappear quickly. The chief symptoms are profound depression, loss of appetite, headache, and insomnia.

Prognosis.—Favorable, so far as the individual attacks are concerned, but poor as to permanent recovery.

Treatment.—The favorable effects of opium not so effective as in ordinary melancholia.

ALTERNATING INSANITY.

Definition.—Circular insanity is a form of insanity in which states of mania and melancholia alternate with each other with or without lucid intervals intervening.

The **symptoms** begin with mania or melancholia. The duration of the cycle may last weeks, months, or years. In some cases there may be marked delusions. The maniacal stage is usually one of simple exaltation.

Prognosis.—Unfavorable. The duration of the disease is for life. Dementia occurs only in advanced stages.

Treatment is unsatisfactory. Chloral fails to quiet. Opium is usually of little benefit during the stage of depression. When possible, rest in bed should be enforced.

DIPSOMANIA.

Definition.—A morbid irresistible desire for intoxicating liquors. The impulse that drives the subject to drink is due to an inherited neuropathic tendency which is too strong to be resisted when the opportunity to indulgence offers.

There is no term expressing the craving for all kinds of stimulants and sedatives, as well as alcohol, so the opium eater, the hashish drinker, the confirmed tobacco smoker are all in the same category. The morbid craving for alcohol is common, and individuals afflicted with this mania will sacrifice their health, their honor, their family, and all that is dear to them to satisfy their longing.

Dipsomania may be **due to** hereditary alcoholism, or to the excessive use of alcohol in childhood, to high-strung nervous temperament, to mental weakness affecting the faculty of volition, to injuries of the head, sunstroke and diseases of the brain, to weakness following long-standing diseases, to neurotic disturbances during the catamenic periods, exhausting employments, the want of all social stimulants or educational development, and senile degeneration of the brain.

Treatment is symptomatic. Seclusion, withdrawal of alco-

hol, and in the event of delirium tremens hypnotics, bed-rest, and food, is all that can be done.

A dipsomaniac should be managed precisely as an insane individual, and as a deprivation from stimulants seems to restore the normal equilibrium, such individuals should be compelled to submit to treatment in sanitariums.

The treatment in dipsomania is usually begun too late. If there be the slightest hereditary tendency in that direction, treatment should be commenced in childhood. The child should lead an outdoor life away from the city, and be trained in strictest morality. In confirmed dipsomaniacs moral treatment is of very little effect, and too little is known of the various "cures" and the percentage of cures and relapses to allow us to form proper judgment of their value. It may be stated as an axiom that if a dipsomaniac does not want to be cured no power on earth can cure him.

Prognosis.—Unfavorable. A drunkard may reform, but the dipsomaniac is never cured of his morbid appetite.

QUESTIONS.

What is recurrent mania?
What are the symptoms?
What is the prognosis?
What is the treatment?
What is recurrent melancholia?
What are the symptoms?
What is the prognosis?
What is the treatment?
What is circular insanity?
What are the symptoms?
What is the prognosis?
What is the diagnosis?
What is dipsomania?
What is the prognosis?
What is the treatment?

CHAPTER V.

INSANITY DUE TO SIMPLE DISTURBANCE OF NUTRITION (ANÆMIA AND HYPERÆMIA) OF THE BRAIN.

MELANCHOLIA.

Definition.—Melancholia is a mental disturbance characterized by a spontaneous, painful emotional state with depression of all the mental and nervous functions and very frequently associated with suicidal tendencies.

Symptoms.—It is difficult to make a distinction between "lowness of spirits," "depression of mind," melancholy or hypochondriasis and pathologic melancholia, as one seems to merge into the other by fine gradations.

The general distinction between a melancholiac and a hypochondriac may be said to be that the depression of the melancholiac is mainly mental, relating chiefly to subjects in harmony with the mind of the patient, while that of the hypochondriac relates primarily to supposed bodily conditions.

Varieties.—Melancholia occurs in many forms, each one with its distinct physiologic and clinical symptoms. Some authors group melancholia according to the most pronounced symptoms; therefore, apart from simple melancholia, we have suicidal and homicidal melancholia, organic (due to organic brain disease), epileptiform or convulsive, resistive, excited, delusional, and hypochondriacal melancholia.

Symptoms of Simple Melancholia.—The patient is noticeably dejected, having a sense of unexplained depression, a vague fear of some impending trouble, a gloom overshadows every one of the patient's actions. He is conscious of this depression, but can not rid himself of it. He gradually begins to lose his interest in business affairs, neglects his friends or family, worries about past mistakes, and constantly reviews his past actions, and entertains fears for the future. The simplest business matters become troublesome, and the most trivial occurrences are magnified. All exertion is avoided, the gait becomes shuffling and slow, the patient appears down-

cast, and desires to be left alone. The appetite becomes dull, sexual desires lessen, the bowels are costive, skin dry, the sleep disturbed. There is distinct loss of weight. The face expresses discontent and misery, and is usually pale and sallow; the eye loses its vivacity; the tongue is coated; the urine is scanty; the pupils are unusually dilated. In women, menstruation is suspended. Many patients complain of dull headaches, and all symptoms of neurasthenia actively appear. These attacks may last several months without mental disturbances, and the chief danger seems to be suicide, which is more apt to be resorted to in the early stages than in the later ones.

Symptoms of Hypochondriacal Melancholia.—The hypochondriac begins to worry about his physical condition. He constantly watches his stomach and his body, and exaggerates every abnormal condition. He watches the urine and excrements, and the slightest attack of indigestion or palpitation is magnified into serious disturbances of the stomach or heart. He consults one physician after another. His will-power is weakened; he becomes vacillating. Hallucinations and delusions may develop, and the symptoms are more decided and positive than those of simple melancholia. While the radical habits of life are not entirely perverted, self-control is partially lost. Every mental pain is associated by the patient with some disorder of the bodily organs or functions. A sense of ill-being is substituted for the healthy pleasure of living, and this ill-being is referred to some organ or function of the body. There are no limits to the fancies of the hypochondriac; but while the simple hypochondriac can do his work, and has a fair amount of self-control, has the power to stop talking of his ailments when he wishes, and does no harm to himself, the patient suffering from hypochondria can not throw off his mental depression; he can not work; he outrages decency openly; threatens to commit suicide, and can not free his mind of his delusions.

From the beginning, in **delusional melancholia, the most prominent symptoms** are delusions, and these delusions are always of the same character (fixed delusions). These delu-

sions are so marked that the patient attributes his disease to the delusions, and regards the depression as secondary. In the majority of cases the delusions and the depression are both caused by a disorder of the brain commonly developed by a hereditary tendency, and brought on by some other disease in some other part of the body, or by unnatural conditions of life. Sense-disturbances appear to excite the apprehension of the patient, and the depression is more profound. Somnambulism and restlessness and agitation of the nervous system result, undermining the general health very rapidly, and causing an active suicidal tendency. It is impossible to hold the attention of the patient, as he is so absorbed with his delusions. He accuses himself of having committed great crimes for which he is to receive terrible punishment. The patient may also show homicidal tendency in order, as he states, to save his family and himself from greater suffering. The organic functions and appetites are interfered with. Nourishment is refused, and digestion and alimentation are changed to a feeling of pain, which pain is frequently very severe. In some instances one finds that actual physical ailment of the intestinal organs exists, which acts as a primary predisposing cause. Some cases begin as simple melancholia, followed by religious delusions and visceral delusions. The patient insists that he has no œsophagus, refuses food, and gradually starves to death. Others imagine that animal food given is human flesh and poison. The delusions of patients refer to ridiculous, paltry things, while others have intense religious delusions. Most patients show suspicion and fear in their faces. The eyes are fixed; extremities cold; bowels inactive; they press their hands upon their foreheads, pinch their palms, or rub their heads.

In **motor (excited) melancholia** the motor centres are affected to a great extent. The patients rush about violently, walk up and down like caged animals, weep and groan, tear their clothes, and their muscular expression constantly changes. After a time the motor symptoms become so prominent that the patient appears at times to be in convulsions although these are not true convulsions. If the symptoms are very

marked, the progress of this form of melancholia is very rapid, and even after temporary improvement relapses are of frequent occurrence. In boys and in girls melancholia is usually of the motor kind, especially accompanied with weeping.

In **resistive (obstinate) melancholia** resistance of a passive or active nature is a marked symptom. The patient objects to dress, or to undress, to go to bed, to move about, to urinate, and so on, and this obstinacy is at times so marked that a great deal of force has to be used to overcome it, and when great force is used an aggravation of the patient's mental condition takes place, causing excitement and even violence. The patient can not explain why he is so obstinate, but is so persistently and unreasonably. This resistance is sometimes accompanied by motor excitement, and is a direct result of certain forms of delusion. The patient imagines that he can not afford to wear clothes, and therefore refuses to put them on, or imagines that the ground on which he stands is unsteady, and therefore he will not move. Masturbation often causes, aggravates, or accompanies this condition.

In **convulsive (epileptiform) melancholia** the motor excitement is a true convulsion accompanied with unconsciousness, which convulsion may occur once or twice in an attack, and differing but little from an ordinary epileptic attack. This form of melancholia is very rare, and is also one of the most serious to deal with. The melancholia is very intense, accompanied by extreme muscular excitement, great obstinacy, and insensibility to pain. These cases are practically incurable.

In **organic melancholia** the lesions are the result of an organic brain disease, and the symptoms usually manifest themselves during the first part of the brain disease. The patient has difficulty in coming to quick decisions. He loses his mental energies, and can not do his usual work properly. His memory is lost and melancholia of a simple type presents itself. It usually ends in dementia as the brain disease progresses.

In **suicidal** and **homicidal melancholia** the tendency to commit suicide or homicide is a most prominent symptom.

This intention may be brought about suddenly by a suggestion from without or within. This tendency to suicide or homicide should always be guarded against by the physician. The tendency to suicide may come about suddenly after calm reasoning, or from motives to escape torture, usually imaginary, or there may be a direct delusion leading to suicide. Some patients fear death, but have a morbid impulse to anticipate it. Others attempt suicide in a state of false consciousness with no memory of the act afterward. As a rule the more a patient speaks of suicide the less he is apt to commit it, but we should never relinquish our vigilance against it. The greatest danger of suicide is near the commencement of the disease, as the impulse is then the strongest. The homicidal feeling may be coexistent with the suicidal, but is much rarer.

Causation.—Anything depressing the general nutrition in one predisposed to insanity may cause melancholia.

In about a half of all cases there is a psychopathic ancestry.

Diagnosis.—In many forms of insanity psychical depression is a stage in the development of the disorder.

In true melancholia every emotion, thought, and act is dominated by the sense of profound depression.

Prognosis.—Generally favorable under appropriate treatment. From 75 to 80 per cent. of cases should recover with proper care. The symptoms usually begin gradually, and should be treated at the start.

Treatment.—In cases of melancholia home treatment is often practicable if a good attendant with tact and firmness is secured; but even in these favorable circumstances treatment in an institution is always preferable. Refusal of food and medicine must be met with positiveness, and in case of resistance forcible feeding must be practised.

As refusal of food is sometimes due to gastric or intestinal disorders, the patient should always be carefully examined to determine whether the gastroïntestinal canal is in normal condition. Concentrated or partially digested foods, such as beef-juice, clam-juice, peptones, etc., may be employed with benefit.

Nux vomica or strychnine, quinine, phosphorus, or cod-liver-oil will often be found of use.

Constipation should be counteracted by the nightly administration of compound licorice powder, cascara sagrada, or one of the usual anticonstipation pills.

Perhaps the most important remedy in acute melancholia is rest in bed.

If an hypnotic is necessary, morphine, sulphonal, or paraldehyde may be used, when absolutely necessary, but a pint of ale or beer, or a glass of whisky and water is often of greater benefit than the medicines mentioned.

The tendency to suicide in melancholia requires careful and constant watchfulness.

The medicinal agent of most value is opium. It is best given in the form of deodorized tincture diluted with whisky and combined with a laxative, as cascara, to diminish the constipating effects of the remedy.

Laudanum should be given in progressive doses, commencing with five drops and increasing five drops each day until distinct improvement in the patient's condition is observed.

Tincture of nux vomica is given in small doses.

MANIA.

Definition.—Mania is a spontaneous exalted emotional state with increased activity of all the mental and nervous functions.

Mania is **characterized** by an abnormal exaltation and activity of the mental functions. It usually manifests itself by irregular talking and acting, by illusions, hallucinations, delusions, and by unusual activity of muscular movements. Like conditions of mental depression, states of mental exaltation up to a certain degree may be normal and physiologic, especially in persons of sanguine temperament; but if such mental excitement occurs without sufficient cause, or it persists long after the cause has operated, then we are apt to consider the person thus afflicted to be subject to mania. *Mental* exaltation is a physiologic state in childhood, which

particularly manifests itself during febrile disorders of children. In adults of a neurotic tendency, blood-poisoning, alcohol, and various drugs readily cause mental exaltation, which exaltation in many instances becomes a prolonged attack of mania.

Symptoms in General.—Beginning gradually, usually with prodromata of depression, occasionally sudden in onset, as after childbirth, the symptoms at first are those of melancholia, gradually passing into a state of exhilaration, with keen perception, great activity, clear consciousness, a sense of power, an increase of all physical functions, constant activity and consequent loss of sleep. The torrent of ideas and impulses becomes greater, to which the patient yields without restraint. Opposition causes irritability, acts of violence, profane language, and mania with delusions and frenzy.

Varieties.—Mania may be simple, subacute, delirious, delusional, chronic, transitory, homicidal, etc.

In **simple mania** a person usually noted for his wisdom, morality, religion, etc., suddenly undergoes a total change, becoming rash, careless, foolish, and doubtfully moral. Such persons may be legally sane, as they do not lose their self-control entirely and act manifestly as lunatics. The great difficulty in such cases is that the patients do not know there is anything wrong with them, and will not believe it. They may look well, but do not sleep well. They lose their finer feelings, and eventually show symptoms of undue exaltation of the sexual desire, and a disregard of all propriety in gratifying it.

Patients with simple mania always think themselves in the right. They exhibit a morbid vanity, or they show a lack of controlling power, not so much in speech as in want of muscular inhibition. Simple mania is often the first stage of acute mania.

Acute mania usually begins quite suddenly. There is a history of physical or mental overwork and continued mental application without rest. Sometimes there is a history of reverses or successes in business, of physical exhaustion, or of alcoholic or sexual excess. It often begins with melan-

cholia, or with certain delusions. The onset is rarely sudden, there always being premonitory signs, producing a profound depression upon an individual possessing neurotic tendencies. The appetite is lost, the patient loses weight, and complains of persistent insomnia with an ill-defined sense of apprehension. There may be disturbances in the circulation, especially located in the brain. Sometimes the patient becomes very restless, and much irritated, the ideas are uncontrollable, there is a tendency to constant talking, with incoherence of speech. The patient's manner changes. He may continually laugh, scold, or swear; conversations are held with imaginary people and hallucinations of smell and touch manifest themselves. Scenes of long-forgotten subjects will be vividly recollected, and self-control is utterly lost. Ideas originate with extreme rapidity.

Delirious mania is a still further stage of acute mania in which the patient becomes actively excited, mistakes his identity, and would readily injure himself or those near him. His speech becomes incoherent, and there is no consciousness of the calls of nature. Delirium appears suddenly and is active and profound from the onset.

The patient is so profoundly absorbed in his delusions and hallucinations that he pays no attention to those about him, and these delusions usually excite his terror and apprehension. Insomnia is marked and prolonged. There may be a sudden return of reason followed very soon by a return of maniacal excitement. During the paroxysm there are great motor activity, fever, rapid pulse, a heavily coated tongue, followed as a rule by symptoms of extreme exhaustion, terminating frequently in death within a short time.

In delirious mania the symptoms are analogous to delirious melancholia except that the patient exhibits maniacal instead of melancholic symptoms. This form may be called delusional insanity plus the maniacal conduct. If the fixed delusions persist for a long time, the maniacal symptoms gradually increase and become permanent.

Chronic mania is a form of mania which usually follows the acute form after the acute symptoms subside, leaving the

mental faculties permanently damaged. In these cases invariably we find a change in the nutrition and blood-supply of the brain.

Chronic mania does not necessarily mean incurable mania. The patient may resume his former habits and mode of living, but does not return to his usual mental equilibrium, showing some mental weakness. The patients with chronic mania sleep very little and symptoms of dementia gradually supervene. The memory becomes impaired. The patient can not be roused to his former habits and instincts. He may lose his affection for those near and dear to him. The patient may have a sudden recurrence of all the active symptoms (**paroxysmal mania**), and there is a tendency to a continued mental derangement. The symptoms may persist for a number of years and all traces of orderly behavior become obliterated.

In **transitory mania** (**ephemeral mania**) symptoms of exaltation come on very suddenly accompanied by incoherence, partial or complete lapse of memory, and sleepiness. The attacks may last a few minutes or a few days, and are frequently epileptiform in character. The febrile inflammatory diseases, erysipelas, typhus fever, etc., may act as predisposing causes. The symptoms in these cases usually end as suddenly as they appear, and there may never be any recurrence.

Homicidal mania is a term applied to the homicidal impulse which manifests itself in the various forms of mental disease. But there are cases of pure homicidal mania in which the homicidal impulse is not accompanied by depression or exaltation of the mind. This form of mania may be due to delusions, to excess of motor energy, to morbid impulses to kill, or to delirium of illness.

Course.—An attack of mania may terminate—(1) in recovery; (2) death from exhaustion; (3) chronic mania; (4) consecutive dementia.

Diagnosis.—The diagnosis of the **clinical** **of mental** disturbance termed mania **is sometimes** **except** for the history of the case.

Causation.—Heredity, prolonged excitement of the cerebral centres, overwork, and mental strain of various kinds may be regarded as etiologic factors.

Prognosis.—Recovery occurs in about 70 per cent. of cases. About 8 to 10 per cent. of cases of mania die from exhaustion. Recurrence is not frequent in cases that have been cured.

Treatment.—Home treatment is generally impracticable. It is customary in most hospitals for the insane to isolate the maniacal patient. Keep the patient in an open ward, preferably in bed, in the presence of other patients, constantly suggesting to him that he is sick and requires treatment and by these means you will soon quiet the most excitable maniac. A bath, clean linen, and quiet, tactful nursing will do wonders in calming the excitement and dissipating the delusions of the maniac.

A useful preliminary is a large rectal lavement, to remove fecal accumulations and prevent soiling of the bed. Feeding with nutritious food is of the first importance. When the pulse is weak and rapid, a moderate quantity of alcohol is useful. Some hypnotic must be given. Among the hypnotics least likely to disturb digestion or depress the appetite are bromide of potassium, chloral, hyoscine, sulphonal, and trional. Digitalis, strophanthus, or strychnine may be added to the bromide-and-chloral mixture. Paraldehyde is a valuable hypnotic in cases with depression.

Opium is generally contraindicated in mania.

Duboisine sulphate, by the mouth, and also hypodermatically in doses of $\frac{1}{100}$ to $\frac{1}{32}$ grain, may be used in cases of excitement.

Hydrobromide of hyoscine is preferred to the hydrochloride, and should be given hypodermatically.

All sources of irritation should be removed from the patient; visits of friends should not be allowed too soon or too frequently.

QUESTIONS.

What is melancholia?
What are the physical and mental symptoms of melancholia?
What is the etiology of melancholia?
What is the differential diagnosis of melancholia?

What is the prognosis of melancholia?
What is the treatment of melancholia?
What is mania?
Give the symptoms of mania?
Give the etiology of mania?
What are the diagnosis and prognosis of mania?
What is the treatment of mania?

CHAPTER VI.

INSANITY DUE TO MICROSCOPIC STRUCTURAL ALTERATIONS IN THE BRAIN.

GENERAL PARESIS.

Synonyms.—Paretic dementia; General paralysis of the insane; Dementia paralytica; Softening of the brain.

Definition.—General paresis is a chronic, progressive, diffuse encephalitis resulting in structural alteration in the cerebral tissue, with involvement of the cortical and meningeal blood- and lymph-vessels, presenting characteristic psychical, motor, vasomotor, and sensory symptoms, with progressive course and fatal termination, usually within three years.

The **course of paresis** may be divided into **three stages**: First, the period of incubation (the prodromal stage). Second, a stage of pronounced monomaniac activity with symptoms of paralysis. Third, stage of extreme enfeeblement with diminution and final loss of physical power.

There is no distinct line of demarcation between these stages as one merges into the other. The symptoms of the prodromal stage are usually so indistinct that neither friends nor family take notice of the patient's condition until some outrageous act is committed. In typical cases the first stage is that of fibrillary tremblings and slight incoördination of the muscles of speech and facial expression, and of mental exaltation and excitement; the second stage is that of muscular coördination and par[illegible]s with mental enfeeblement;

and the third stage is marked by advanced paralysis, almost inarticulate speech, ending with general paralysis and mental extinction.

Symptoms.—Paresis begins very gradually, with symptoms characteristic of neurasthenia or of dyspepsia.

Among the early psychical symptoms are irritability, and especially an instability of the moral and mental character. Memory becomes defective. The patient's affairs are in confusion. The moral sense is often perverted. He becomes unconventional, uses bad language, neglects his family, consorts with drunkards and lewd females, makes indecent proposals to respectable women of his acquaintance, all this without recognizing any impropriety in it.

The patient can not concentrate the attention; speech is a little thick, indistinct and hesitating, syllables are dropped or repeated, speech finally becoming an inarticulate sound.

The prevailing character of the psychical manifestations is one of exaltation. Delusions of persecution may be present, and are generally attended by expansive delusions.

Delusions of grandeur are usual in most cases of general paresis. Many cases of general paresis run their entire course without manifesting exaltation or expansive delusions at any time. The delusions are rarely fixed.

As the disease progresses, dementia becomes more and more marked.

One of the earliest physical symptoms is persistent insomnia, often accompanied by intense and frequently recurring hemicrania. In other cases there is an uncontrollable desire to sleep.

Early symptoms also are recurring attacks of loss of consciousness. They are present in nearly every case and are an important diagnostic sign. After severe attacks there may be hemiplegia, which, however, usually disappears as a rule in a few hours or days.

Epileptiform convulsions, may also be present as early symptoms, but are usually met with in the later stages.

The pupil is irregular, mostly dilated, rarely contracted. The pupils are often unequal and react slowly to light.

Sensation may be retarded or entirely abolished. The Argyll-Robertson pupil, so characteristic of tabes, is also a frequently noticed symptom of general paresis. The patellar reflex is most often increased.

The affection is one of fine coördinated movements—a cortical ataxia, rather than gross paralysis.

A fibrillary tremor or twitching of the muscles about the mouth may be present. On protruding the tongue the organ is tremulous or protruded in a spastic or jerky manner. Tremor of the hands is also present as a symptom of the advanced stage. The speech is jerky and slow. In advanced cases it becomes slurring. Syllables are dropped or repeated. Certain words are pronounced with difficulty. The speech finally degenerates into an inarticulate sound.

An early symptom is retention of urine. An annoying symptom of cortical irritation is a constant grinding of the teeth. The gait in the early stages is spastic or ataxic; in advanced cases it becomes slouching or dragging. Vasomotor disturbances are frequent. Redness or blueness of the skin, œdema and cyanosis of the peripheral members, and diminution of blood-pressure are due to vasomotor disturbances. Trophic changes occur and liability to bedsores and to cystitis is great. The course of general paresis is, as a rule, steadily progressive.

When paretic dementia is fully developed, the mind does not perceive accurately anything. Illusions and hallucinations occur, followed by marked loss of power of sensory perception. The emotional state is unstable, but rarely shows any tendency to suicide. Consciousness becomes very imperfect, ending finally in complete suspension of mental action.

Some cases are complicated by posterior or lateral spinal sclerosis and in rare cases paresis follows sclerosis.

Diagnosis.—In **syphilis** there are more frequently symptoms referable to gross brain-lesions. **Tabes** has strongly marked motor and sensory symptoms.

Chronic alcoholism sometimes presents symptoms resembling early general paresis, but the ocular symptoms of the latter are absent.

In **neurasthenia** delusions of grandeur do not occur, and, in place of the feeling of well-being as expressed by the paretic, the most minute details of physical symptoms are given. **Chronic pachymeningitis** or **multiple cerebral sclerosis** resembles paresis.

Causation.—Paresis rarely begins before the thirty-fifth and still more rarely after the fiftieth year of life. It attacks by preference persons in the higher walks of life. Syphilis is regarded by many authorities as the most common single cause. Mental stress, especially when associated with intemperance, venereal excesses, or other irregular habits are also etiologic factors.

It is more frequent in cities than in country-districts. Men are attacked from three to five times as often as women.

Pathologic Anatomy.—General paresis is a psychosis based upon recognizable structural alterations in the brain. The vascular sheaths are filled with white and red blood-corpuscles, the vascular walls thickened, and the calibre of the vessels diminished. In the substance of the brain there is an increase of the connective-tissue elements which, there is reason to believe, produce atrophy of the brain-cells by pressure. The entire brain takes part in this process.

In the cerebral tissue there are a degeneration of the finest nerve-fibrillæ, and also a swelling, hyaline or fatty degeneration, vacuolization, pigmentation, and finally atrophy of the cell-bodies.

The arachnopia is generally cloudy and thickened. The convolutions are diminished in volume and the fissures wider than normal. The cortical substance is decreased. The average diminution in weight of the brain amounts to 100 to 200 grammes (3 to 6 ounces).

There is a widely-pervading cell-degeneration of a granular, probably fatty type; overgrowth of the connective-tissue structure within the cerebral substance, and a diffuse, inflammatory change around the sheaths of the vessels with slighter alterations in the sheaths themselves. The skull is at times markedly thickened. In the medulla oblongata and the

spinal cord structural alterations similar to those in the brain are found.

Prognosis.—The general experience is that general paresis is incurable. It may be of short duration, but usually lasts three years; occasionally terminates in five or six years.

Treatment.—Complete rest from business and removal from all sources of irritation are the first object to be striven for. Dissipation, intemperance, and venereal excesses must be abandoned. Removal to a properly managed institution as early as practicable is, therefore, to be urged. Ergot should be used continuously.

Mercury and the iodides, the latter in large doses, may cause arrest of the connective-tissue proliferation, and the absorption of the new formation in the brain and spinal cord. From ½ to 1 ounce of iodide should be given daily, beginning with moderate doses and ascending by a stated daily increment until the patient's tolerance is reached.

For sleeplessness, chloral, bromides, sulphonal, and paraldehyde are indicated.

Great care is necessary in feeding advanced cases to prevent bolting of large morsels of food, and consequent asphyxia from entrance of food into or compression of the air-passages.

Cleanliness and frequent changes of position in those patients who have become bedfast from the advance of paralytic symptoms will prevent bedsores.

KATATONIA.

Katatonia is a form of insanity **characterized by** depression, exaltation, stupor, confusion, and dementia, usually occurring in regular cyclic sequence. Occasionally there is a tendency to rhythmic movements of muscles.

The **symptoms** begin gradually after great exhaustion or suddenly after emotional shock. The patient sinks into a state of indifference and acts as if in semistupor. Occasionally he passes into a state of ecstasy or catalepsy. Attempts at suicide are occasionally made. Patients may refuse food, are restless and in a state of exaltation varying with depres-

sion. Delusions of grandeur, or fits of rage culminating in attacks upon bystanders or in destructive tendencies, are common.

Mutism, or dumb stupor, is usually present as a stage in the course of the disease. The patient sits or stands in one position, with head and eyes down, and apparently taking notice of nothing passing around him.

Cataleptic seizures are not infrequent.

There are othæmatoma, often anæsthesia, disturbances of nutrition, and loss of control over the sphincters.

There is a peculiar aberration of speech, termed by Kahlbaum "verbigeration," and a rhythmic or stereotypic movement of certain groups of muscles. A spastic pouting called "Schnauzkrampf" by the German authorities is described as especially frequent.

Verbigeration is a rhythmic reproduction of sounds, words, or sentences, often without logical connection, which are repeated in a declamatory or pathetic style.

In advanced cases consecutive dementia comes on.

Etiology.—A neuropathic constitution, hereditary or acquired, is a predisposing factor. Masturbation is mentioned by authors as a frequent factor.

Diagnosis.—Stereotypic movements, muscular tension, and stupor are observed in other mental disorders, especially in acute dementia, paranoia, general paresis, acute hallucinatory delirium, grave hysteric conditions, and the insanities of pregnancy, of the puerperal period, and of lactation.

Prognosis is unfavorable. Recovery rarely occurs.

Treatment is essentially symptomatic.

CONSECUTIVE DEMENTIA.

Synonyms.—Terminal dementia; Blödsinn.

Definition.—Consecutive dementia is a state of permanent and incurable weakmindedness following an acute psychosis. It is the final result of all forms of insanity which become chronic.

Symptoms and Course.—This form of dementia usually fol-

lows melancholia and the various forms of mania, epilepsy, or general paralysis. After a prolonged period of depression or exaltation a stage of quiet and repose follows; the violence of the shock of the disease having expended itself, the brain remains damaged. General nutrition may have been restored, the body may have increased in weight, but there is no mental improvement, the patient having settled into a passive, indifferent stage; the memory has become weakened, natural affection, power of attention and concentration have disappeared. The patient becomes indifferent to his appearance, knows no emotion, has no fixed delusions, and does not respond to any impulse.

The subject may recover sufficiently from the acute psychosis to perform properly mechanical labor of various sorts, but consecutive thought, especially upon a complex subject, is impossible. Consecutive action, as well as consecutive thinking, becomes impossible.

There is also a loss of motive power.

Speech is defective and there is mnemonic aphasia.

Diagnosis.—Consecutive dementia resembles in many respects idiocy and imbecility, from which it is differentiated by the history.

Prognosis.—Consecutive dementia, being due to structural alteration in the brain-tissue, is incurable.

Treatment.—This is purely symptomatic.

SENILE DEMENTIA.

Definition.—Senile dementia is a chronic, progressive weak-mindedness due to structural alteration in the brain occurring in advanced life.

Etiology.—Senile dementia is rare before the sixtieth year. Its course is usually slow, running over several years.

Symptoms.—It is impossible to give the psychology of normal old age— that is, of the normal physiologic abatement and decay of the mental functions as a result of the brain shrinkage and the lessening of energy and of all other functions of the organism.

Physiologic senility means a loss of reproductive power, a diminished effective faculty, lessening power of attention and memory, diminished desire and power to perform mental and bodily functions, a lowered imagination and enthusiasm, diminished adaptability to change, greater slowness of mental action, slower speech, impaired muscular coördination, a change in the tone of voice, a diminution of blood-corpuscles, a lessening in bulk of the body, especially of the brain, which becomes altered structurally and chemically.

The dangers of normal senility are hereditary brain weakness, a diseased vascular system, and overexertion of the brain during former periods of life.

It is impossible to fix an age when senility begins. Some races grow old early. Imbeciles, idiots, and many of the insane begin to show signs of decay at an early age.

As the physical powers decay with advancing years, the intellectual functions also become imperfect.

Memory of recent occurrences is impaired, but the recollection of past events is often vivid. The subject becomes suspicious of his relatives and friends, is easily excited and irritated, and frequently misplaces articles of ordinary use in life.

Among the prominent symptoms are increased sexual desire, with diminution of power to perform the sexual act.

The senile dement is obstinate and vain.

The speech is halting; the gait becomes shuffling; there is loss of control over the sphincters, and occasional slight paralytic strokes. The sleep is usually disturbed, and there is often a distinct tendency to stray away.

Pathology.—Patients dying of senile insanity show the grossest pathologic changes in the brain, and most common of all lesions is that form which may be termed "softening of the brain," a localized necrosis, partial or entire, of a portion of brain-tissue, resulting in most cases from a deprivation of blood from embolism or thrombosis of the arterial branches supplying it. The softening is usually localized and is seldom extensive, differing in this respect from a softening of younger insane people. The most common sites are a

softening of the basal ganglia, the convolutions of the vertex, and the lateral portions of the anterior lobes. The necrosis is more marked in the white tissues than in the gray. The whole brain appears atrophied, the skull-cap is thickened, especially over the anterior lobes, the dura mater adheres to the skull-cap, and the pia mater is thick and fibrous.

Diagnosis.—The history of the case is the best diagnostic symptom.

Prognosis is unfavorable.

Treatment.—The treatment is symptomatic.

When there is defective circulation a mild stimulant may be useful. Sleep is best induced by malt liquors, paraldehyde, trional, or opium.

EPILEPTIC DEMENTIA.

Definition.—Epileptic dementia is a form occurring in advanced stages of epilepsy, due to structural alterations in brain-tissue.

Etiology.—In a considerable proportion of cases epilepsy is accompanied with mental disturbances, frequently ending in insanity. The frequent recurrence of epileptic fits has a tendency to impair the mental faculties. Epileptic insanity rarely follows the first appearance of the fits, and the more severe and the more frequent the fits the greater is the risk of insanity. This form of insanity may follow after attacks, usually within twenty-four hours, or before the attacks, a day or two before the seizure, or mental disturbance may occur instead of the attack, or the fits may disappear entirely, and some form of chronic insanity may take their place.

There are two epileptogenetic periods—the first during the period of the fastest brain growth from birth to the age of seven, and the next during the period of puberty and early activity of the reproductive system from thirteen to eighteen.

Epilepsy is not always a primary cause of the insanity, but it may appear during the course of chronic insanity.

The chief **symptoms** associated with or caused by epileptic fits are irritability and impulsiveness. In many cases the

maniacal condition is more pronounced than in any other form of insanity, and this condition is more frequently the cause of murder than even the alcoholic form. Some epileptics have a distinct readiness to emotional mania. Epileptic insanity appears in many varieties, just as there are many varieties of epilepsy itself. Some patients show irritability; others have maniacal actions, with or without consciousness; some show impulsive actions; other exhibit a distinct stupor, depression, false consciousness, automatism, an epileptic want of realization of the dangers to which they are exposed during fits, and marked sensory neuroses.

Prognosis.—Most patients show distinct improvement of their mental symptoms when the epilepsy itself is treated with remedies much in vogue at present.

Most patients die in *status epilepticus*, complicating pneumonia, or exhaustion.

Treatment.—The usual remedies for epilepsy may delay the progress of the dementia, but they can not arrest it.

QUESTIONS.

What is general paresis?
Give the etiology of paresis.
Give the symptoms and course of paresis.
What are the diagnosis and prognosis of paresis?
What is the treatment of paresis?
What is katatonia?
What is the etiology of katatonia?
What are the symptoms of katatonia?
Give the prognosis and treatment of katatonia.
What is consecutive dementia?
What are the symptoms of consecutive dementia?
What are the diagnosis, prognosis, and treatment of consecutive dementia?
Define senile dementia.
What are the symptoms of senile dementia?
What are the etiology and diagnosis of senile dementia?
What are the prognosis and treatment of senile dementia?
What is epileptic dementia?
What are the symptoms and treatment of epileptic dementia?

CHAPTER VII.

INSANITY DUE TO ORGANIC CHANGES IN THE BRAIN.

SYPHILITIC INSANITY.

Definition.—Insanity due to syphilitic new formations in the brain or meninges.

General Symptoms.—Severe and long-continued headache, more intense usually at night. Attacks of unconsciousness, sometimes convulsions and coma. After one of these attacks there is frequently local or general paralysis, which may be transitory or permanent.

Stupor and depression may alternate with maniacal outbreaks. The memory is often profoundly impaired.

The **symptoms** vary greatly as the functions affected are numerous, and because the effects of the disease are often slight and slow in development, and multifarious in kind. The character of the symptoms usually points to the position and intensity of the pathologic changes in the brain.

Some authors for convenience' sake distinguish **four forms of syphilitic insanity**—

The first may be termed **secondary syphilitic insanity.** It occurs during the second stage of the disease when the eruption appears, is curable and rare. There are marked mental excitement, an extreme amount of motor restlessness, and at times delirium. This excitement gradually diminishes, coinciding with the gradual disappearance of the syphilide. These maniacal attacks may occur with each new symptom of the syphilitic disease, and with the disappearance of all syphilitic symptoms the mental state becomes normal. This form of syphilitic insanity has no known pathology.

The second form may be termed **delusional syphilitic insanity**, caused by slight starvation from an obscure syphilitic irritation that has become arrested. Its main symptoms are monomania of suspicion, sensory perversion, hallucina-

tions of the senses. Under the influence of delusions the patient may become dangerous. This form of insanity is rare, and almost always incurable.

The third form may be termed **vascular syphilitic insanity** where the syphilitic poison affects the bloodvessels of the brain, causing slowly developing arteritis and consequently slow elevation of the cerebral tissue. The patient becomes morbidly irritable, disregards the decencies of life and social status, and a steady deterioration of the mental powers takes place. At times there are convulsive symptoms and partial paralysis. Memory becomes gradually lost. Stupor, with other signs of greatly damaged brain-function, follows, and finally death ensues. The symptoms vary according to the number and location of the arteries affected. The duration is various in different cases, but it is rarely less than five years, and in some cases it may be twenty-five. If the lumen of an artery has been diminished, there is very little hope that therapeutic means can ever restore it.

The fourth form may be termed **true syphilomatous insanity.** This may consist of a syphilitic meningitis characterized by convulsions, temporary stupor and delirium, or there may be a quickly growing syphilitic tumor within a convolution, causing after a few weeks an extensive softening of the brain, with maniacal excitement and convulsions, and may be followed by speedy death, or there may be local gummata, causing, through pressure, local convulsions, mental irritation, and slowly progressing dementia, or there may be great masses of syphilitic material and purulent deposits over one or both hemispheres, causing gradual dementia, and at last coma, or there may be membranous or bony tertiary lesions, setting up a form of epileptic insanity.

In advanced stages the dementia is usually profound.

Diagnosis.—Depends upon the history. A differentiation from general paresis is often impossible.

Pathologic Anatomy.—The syphilitic neoplasm may be in the form of a diffused gummatous meningitis, endarteritis, or gummatous foci in the brain.

Prognosis.—In the early stages if appropriate treatment is promptly instituted the prognosis is not unfavorable.

Treatment.—Mercurial inunction and potassium iodide in large doses should be employed as soon as a probable diagnosis is made. Ferruginous tonics will generally be required.

POSTAPOPLECTIC INSANITY.

Definition.—Insanity following destruction of an area of brain-tissue, due to cerebral hemorrhage or embolism.

Symptoms.—In addition to the usual physical symptoms following brain-lesions there are loss of memory, dementia, and occasional attacks of emotional disturbance or outbreaks of maniacal violence.

Treatment.—This can only be symptomatic. Mental restoration is rare in mild cases.

INSANITY FROM CEREBRAL TUMORS AND ABSCESSES.

In many cases of brain-tumor or brain-abscess no psychical symptoms are present. In others, however, there are loss of memory, apathy, dulness of perception, and occasionally intellectual perversion. Hallucinations and delusions may be present. When the neoplasm encroaches upon the visual sphere, hallucinations of vision may complicate loss of sight.

The symptoms as a rule depend upon the location and extent of the tumor or abscess.

Treatment.—Medicinal treatment is only palliative. Surgical interference may be resorted to.

INSANITY FROM CRANIAL TRAUMATISM AND SUNSTROKE.

Insanity follows cranial injuries much more frequently than is commonly supposed, many of the cases recovering from the acute mental disturbances following shock and inflammation later become permanently insane.

Unfortunately traumatic insanity produces a craving for

stimulants, which naturally aggravates all the symptoms. A person addicted to alcohol, receiving cranial injuries, is very apt to develop symptoms of traumatic insanity. Thus we see that alcohol produces a tendency to this form of insanity, and at the same time traumatic insanity creates a tendency to the use of alcoholic stimulants.

Some patients develop symptoms of ordinary epilepsy of a very slowly progressive type. The epileptic symptoms may not manifest themselves immediately after the injury. In others traumatism seems to act as an exciting cause of insanity, when predisposed to the disease.

Treatment.—In cases of fracture of the skull the recognized surgical procedures are indicated. In contusion, opening of the skull at a point opposite to the site of injury is advisable.

In the secondary dementia following brain-injuries surgery is not advisable.

Sunstroke also causes insanity, and the general mental symptoms of traumatism and sunstroke are apt to be alike. In traumatic insanity the motor symptoms are apt to be pronounced in the forms of general difficulties of speech, general muscular weakness, hæmophilia, or paralysis. The sensory symptoms consist of cephalalgia, vertigo, hallucination, incapacity for exertion either bodily or mentally. The mental symptoms are those of melancholia with tendency to hallucinational or delusional insanity. In these cases a minute quantity of alcohol is apt to produce suicidal or homicidal symptoms.

QUESTIONS.

What is syphilitic insanity?
What are the symptoms of syphilitic insanity?
What are the diagnosis and prognosis of syphilitic insanity?
What is the treatment of syphilitic insanity?
What is postapoplectic insanity?
What are the symptoms and treatment of postapoplectic insanity?
How would you explain the fact that some cases of cerebral neoplasm and abscess are without physical symptoms while others have such symptoms in marked degree?
What are the characters of such physical symptoms in cerebral abscess?
How may injury cause insanity temporarily and permanently?
What treatment may be of value?

CHAPTER VIII.

INSANITY DUE TO TOXIC SUBSTANCES CIRCULATING IN THE BRAIN.

ACUTE CONFUSIONAL INSANITY.

Synonyms.—Amentia; Verwirrheit; Wahnsinn.

Definition.—An acute form of mental disturbance, beginning suddenly or with few prodromes, characterized by an impairment of mental action with confusion of ideas due to the presence of illusions and hallucinations, but without uniform emotional disturbance or suspension of mental action.

Etiology.—The poisonous materials may be absorbed from the intestinal canal, from wounds or septic areas, or may be formed in the blood, tissues, or glands, or introduced from without.

Acute confusional insanity occurs during or after infectious diseases, after surgical operations, in the puerperium, during lactation, after cranial and other traumatisms, neuritis; is caused by the ingestion of alcohol, opium, cocaine, lead, and various drugs; from the inhalation of certain poisonous gases.

Symptoms.—Headache and insomnia may precede the outbreak. Usually, the patients suddenly become excited, talkative, have hallucinations or illusions, which are rarely of an agreeable character. They may see rats, snakes, spots of blood, etc. The visual hallucinations are often like those of delirium tremens or the delirium of fever. Auditory hallucinations may also be present. Sometimes there are delusions of suspicion and persecution, and occasionally delusions of grandeur, but these are transitory. The patient soon becomes incoherent, loses all relation of time and space, does not recognize his surroundings, and confounds his own and others' personality. There may be sudden outbreaks of violence, which sometimes lead to homicidal acts, but his acts depend upon the delusion present.

Temporary lucidity may occur, but is usually transitory. One type of nervous disturbance, when once established, is long continued.

Some patients are excessively loquacious. At times the patient utters new words. At other times there is mutism with muscular rigidity.

There is great motor restlessness and control of sphincters is lost.

There is usually fever, with rapid, and in advanced cases, feeble pulse.

Diagnosis—The differentiation must be made from mania and melancholia.

Many of the cases pronounced "agitated melancholia" and "melancholia with stupor" are cases of confusional insanity.

Prognosis—The prognosis is generally favorable.

Treatment—Rest in bed. Isolation is not necessary.

Nutrition demands constant attention.

The insomnia and delirium may often be overcome by warm baths. Of drugs, opium is preferred. Chloral, hyoscine, and paraldehyde are not recommended. Sulphonal and trional may be cautiously tried. Digitalis, strophanthus, and strychnine are often of great value. The bowels should be kept open and tincture of chloride of iron in large doses is of value.

ALCOHOLIC INSANITY.

Alcohol is one of the most common **causes** of insanity. It generally brings into prominence hereditary and acquired brain-weakness. While the excessive use of alcohol may be the direct cause of producing any of the various known forms of insanity where there is a tendency in the individual toward their production, we also find some distinctive forms of insanity brought about directly by the use of alcohol.

Alcoholic insanity manifests itself in **various forms.** The most typical of these are acute alcoholism (delirium tremens), chronic alcoholism, mania-a-potu, and alcoholic degeneration.

Acute alcoholism is a typical, excited, motor melancholia accompanied by hallucinations of sight, misleading delusions, general depression, suicidal tendency, incoherence of speech, failure of memory, impulsive acts, and in some cases by unconsciousness. The most marked physiologic symptoms are

motor restlessness and motor tremulousness. The temperature is above normal. The appetite is lost, digestion and assimilation are faulty, and consequently there is a rapid loss of weight. Insomnia is always very marked. The attacks as a rule are of short duration, especially if proper treatment is resorted to.

Prognosis.—Most patients are apt to recover from these acute attacks if the stimulants are stopped and they are placed under proper surveillance, either in their own homes or in some institution, but those who have lived on alcohol all their lives may pass from acute attacks into those of stupor and coma, followed by death. In some patients, even after recovery, the hallucinations of hearing, or the suspicion of poisoning may persist. In others, while the patients may have recovered completely from their attacks their mental condition is not so clear as it was previously. In those with hereditary tendency to insanity the attack may be followed by melancholia, mania, or dementia.

Treatment.—Withdrawal of the alcohol, substitution of another stimulant for it, like strychnine, supporting and nutritive measures combined with quiet, rest, and, if needed, restraint, fulfil the usual indications.

In **chronic alcoholism**, where the use of alcohol has been persisted in for a long time, the attack of insanity may be ushered in by convulsions, or there may be a gradual development of the symptoms previously described, but the suspicions and fears persist and the delusions become fixed. There are hallucinations of hearing, rarely of sight. The tendency to homicide is marked. Insomnia, while a frequent symptom, is not so persistent as in the acute attack. Speech is thick, the walk becomes somewhat ataxic, the reflexes are lost. Peripheral neuritis develops in most cases.

Prognosis.—Recovery is very rare, as both the brain-tissue and the spinal cord have degenerated. Even with the best of treatment there may be a temporary improvement, but dementia and peripheral neuritis are apt to bring about a fatal ending.

In **mania-a-potu** the symptoms are extremely acute and of short duration. Those of hereditary tendency to brain trouble are apt to develop it, no matter how little alcohol they absorb. A few glasses of any alcoholic beverage make them delirious, riotous, homicidal, and often unconscious. Many patients suffering from *mania-a-potu* have a hereditary craving for stimulants (dipsomania).

The persistent use of stimulants is apt to produce **alcoholic degeneration.** The expression of face and of eyes change, the mental condition becomes lowered, the self-control weakened. They are not able to stand large amounts of work, and senile dementia is apt to develop at a very early age.

Pathology.—Changes in those who have indulged in alcohol for a long period show themselves in the form of a thickened and adherent dura mater and pia mater. In those who develop dementia the brain is markedly atrophic.

The **treatment** in chronic cases is apt to have very little result. There may be temporary improvement, but the final, fatal result is inevitable.

QUESTIONS.

What is confusional insanity?
What are the symptoms?
What is the etiology?
What are the diagnosis and prognosis?
What is the treatment of confusional insanity?
What is the etiologic value of alcohol in insanity?
What peculiarities of its action can you name?
What are the common forms of alcoholic insanity?
Describe the symptoms of acute alcoholism.
What is the prominent physiologic sign of acute alcoholism?
What may be stated as to the duration, prognosis, and treatment of acute alcoholism?
What are the clinical features of chronic alcoholism?
What disease of the nervous system is very common in chronic alcoholism?
What is mania-a-potu?
Is there a clinical feature of great significance in mania-a-potu?
What is meant by alcoholic degeneration?
What are the pathology, the prognosis, and the treatment of the chronic forms of alcoholism?

CHAPTER IX.

INSANITY DUE TO DEVELOPMENTAL CHANGES IN THE BRAIN.

PUBESCENT INSANITY.

Definition.—Insanity occurring during the pubescent period of life.

The **role of the period of puberty** is a most important physiologic era in the life of man or woman. Before puberty the brain has been growing mainly in bulk. There has been a general psychic likeness between the sexes. The mental development of each sex has been more or less in the same direction. After puberty man's mind develops more in the direction of energizing, and woman's mind in the direction of emotion. These changes naturally do not occur all at once, but take years for their completion. It is during this period that hereditary tendencies begin to show their marked influence. If the conditions of environment are favorable, hereditary tendencies may never develop, as Nature has wisely arranged it that the tendency toward reproducing the normal and healthy type is always the strongest. On the other hand, should there be a strong tendency to abnormality the victims usually die before the age of reproduction, and they are thus incapable of transmitting abnormalities to their offspring.

Pubescent insanity does not occur except in individuals having a strong family tendency toward mental defect, and in such individuals it is caused by some irregularity in the development of the reproductive or menstrual functions.

Symptoms and Course.—Clouston describes pubescent insanity as follows: "The insanity of puberty in both sexes is characterized especially by motor restlessness. Such patients never sit down by night or day and never cease moving. There is noisy and violent action, sometimes irregular movements, or, in the few melancholic forms and melancholic stages of the maniacal cases, cataleptic rigidity. The mental symptoms consist most frequently in a kind of inco-

herent delirium rather than any fixed delusional state. In boys the beginning of an attack is frequently ushered in by a disturbance in the emotional condition—dislike to parents or brothers or sisters expressed in a violent, open way; there is irrational dislike to and avoidance of the opposite sex. The manner of a grown-up man is assumed, and an offensive 'forwardness' of air and demeanor. This soon passes into maniacal delirium, which, however, is not apt to last long. It alternates with periods of sanity and even with short periods of depression."

In those cases which do not recover, a mild form of dementia, resembling imbecility, follows.

Prognosis.—Under appropriate management pubescent insanity is amenable to treatment. About half the cases recover.

Treatment.—The treatment of pubescent insanity should be tonic and reconstructive.

The tendency to sexual excitement and to masturbation should be counteracted in a moral way.

ADOLESCENT INSANITY.

Definition.—Insanity occurring during the period of adolescence.

The **symptoms and course** are the same as those of pubescent insanity, except to a more marked degree. The attack occurs usually between the ages of eighteen and twenty-five, when the function of reproduction has attained its full development, when the bones are firmly consolidated, when there is a perfect assumption of the manly form in the male and of the womanly form in the female. The mental change during this period is very marked. There seems to be an immeasurable distance between puberty and adolescence. The sense of right and wrong, the sense of seriousness and responsibility is awakened for the first time. In weak brains this period is apt to bring on psychologic changes.

Prognosis and Treatment.—If hereditary predisposition to insanity be not too marked, appropriate treatment may produce amelioration, and even an ultimate cure. Every effort must be

made to produce a normal completion of the period of adolescence, and we must endeavor to induce a normal state of health. Active exercise in the open air, shower-baths every morning, athletic games of all sorts, large quantities of milk and ordinary wholesome diet are very effective. Codliver-oil, the hypophosphites, and some bitter tonic may also be used. If the patient begins to gain weight in a few months, prognosis is hopeful.

CLIMACTERIC INSANITY.

Definition.—Insanity occurring during the period of sexual involution in women.

Etiology.—Just as the brains of persons who have a hereditary taint of insanity are apt to be upset by the slow development of the sexual function during the period of puberty and adolescence, so they are apt to show symptoms of mental derangement at the time of the climacteric period. The climacteric period usually shows a mental change in both sexes. Sexual desire becomes diminished, the form and expression gradually change, trophic energy becomes lessened. This period usually lasts from one to three years. The mental symptoms may appear before the onset, during, or after the climacteric period.

Symptoms.—Any of the clinical varieties of mental disorder may be present during the climacteric; but melancholia is most frequent.

Hallucinations of hearing and of smell are frequent. Religious delusions are predominant.

The fear of death, immediately impending or more or less remote, is often present.

Delusions and hallucinations referable to the sexual organs are common, but actual disease of the sexual organs is often present.

Delusions of grandeur are sometimes present.

Suicidal tendencies are frequent.

In some cases the depression leads to the use of alcoholic stimulants.

Prognosis.—Is rather favorable.

Treatment.—The precordial anxiety and palpitation of the heart, if troublesome, will generally yield to moderate doses of Hoffmann's anodyne. For insomnia, paraldehyde is probably the least harmful hypnotic that can be used.

The physical depression indicates good food, fresh air, and tonic medication.

Opium should be given systematically, as recommended in melancholia.

PUERPERAL INSANITY, LACTATIONAL INSANITY, AND INSANITY OF PREGNANCY.

Puerperal insanity, *lactational insanity*, and *insanity of pregnancy* are forms of insanity developing in women at childbirth or during the period of nursing or during pregnancy.

Puerperal insanity occurs within six weeks after childbirth, and is comparatively frequent among those of a neurotic tendency, who have been weakened by the drain on their system during the childbearing period.

Primiparæ and those who have undergone difficult labor are more apt to develop it. It appears as acute mania or melancholia in most cases, with marked suicidal impulses. The patient's manner becomes changed. She does not seem to care for her child, and often, if left to herself, is apt to harm her offspring. The patient is erratic, at times gets violent with a supernatural strength. The body looks weak and emaciated, and we frequently find suppression of the lochia and septic symptoms in the uterus. Recovery in these cases is gradual, and depends on our success in building up the system and removing any septic trouble, if such be the cause of the attack.

Lactational insanity usually develops in women who are anæmic, and have been subjected to prolonged nursing. It is more frequent in those who have had children in close succession, and who have in each instance nursed the child themselves. Most patients belong to the poorer classes.

They become greatly depressed, restless, sleepless, have

delusions, and impairment of the special senses. Recovery is common if the child is weaned and the patient is given the benefit of good food and fresh air.

Insanity during pregnancy is very rare, and it is more apt to occur in women who have married and have become mothers when advanced in life, and consequently fear the conditions of pregnancy and the dangers of childbirth to an inordinate degree.

The patient becomes depressed, loses interest in all things, and is apt to develop acute mania, ending in dementia, but the majority of patients recover after having given birth to their child.

OVARIAN INSANITY; UTERINE INSANITY.

Ovarian insanity (**old maids' insanity**) is a form of insanity apt to occur in old maids, usually of an unprepossessing nature, at the time of the cessation of the ovarian function. Such patients, after having led an upright religious life, develop peculiar passions toward some casual acquaintance, whom they believe to be passionately fond of them, and whom they actually accuse of having ravished them. It is usually a clergyman who is accused of this deed, and every look and every gesture of the supposed lover is misinterpreted. None of these patients ever completely recover from this delusion, but usually pass into a mild form of senile insanity.

Uterine Insanity.—Menstruation creates a tendency in women to a slight irritability of the nervous system, and nervous degenerations like neuralgia, migraine, epilepsy, etc., are apt to become worse at this period. A disturbance in menstruation is a constant danger to the mental stability of some women. The paroxysms of mental disease are usually worse at the time of the menstrual period, especially in acute varieties of insanity.

On the other hand, insanity is very apt to cause a suspension or a disturbance of the **normal menstrual** function, but while there may be an **aggravation of mental** symptoms

at the time of menstruation in those suffering from one of the various forms of insanity, insanity may in some instances be the direct result of a disturbance or suspension of menstruation, and these forms of insanity may be termed *uterine.* In most instances the insanity assumes the form of melancholia. The patient becomes depressed and morbid, forgetful, and sleepless, loses her appetite, weeps without cause, is obstinate, and has hallucinations. But all these symptoms disappear just as soon as the amenorrhœa ceases and the menstrual function is reëstablished. In cases of sudden suppression of the menstruation attacks of maniacal delusions are apt to develop.

The treatment in all such cases is directed toward a toning up of the system and a restoration of the normal menstrual function.

QUESTIONS.

What is pubescent insanity?
What are the symptoms?
What are the prognosis and treatment?
What is adolescent insanity?
What are the symptoms?
What are the prognosis and treatment?
What is climacteric insanity?
What are the symptoms?
What are the prognosis and treatment?
What varieties of insanity are associated with reproduction in women?
What are the causes, symptoms, prognosis, and treatment of puerperal insanity?
What is probably the greatest causative factor in lactational insanity?
How would you manage a case of lactational insanity?
In what period of life does insanity of pregnancy occur, and with what symptoms?
Has the insanity of pregnancy any grave importance?

CHAPTER X.

RARER FORMS OF INSANITY.

Classification.—Apart from the common varieties of mental disease some rarer etiologic varieties are met occurring as complications of other systemic diseases; thus we may have the insanity of *Bright's disease,* of *bronchitis, cardiac*

disease, asthma, oxaluria and *phosphaturia,* of *influenza,* of *myxœdema,* of *exophthalmic goitre,* of *lead poisoning, anœmic insanity, diabetic, metastatic insanity, postfebrile insanity, post-connubial insanity, insanity from the loss of the sense of sight or hearing,* of *surgical operations, the delirium of young children,* and of *somnambulism.*

Insanity of Bright's disease results from uræmic poisoning; manifests itself as half delirium and half mania. It occurs in the chronic form of Bright's disease with contracted kidneys, enlarged heart and a tendency to dropsy. The patient is very restless, has delusions, and tries to injure himself. There are remissions when the patient is quiet and rational, but very much prostrated. With each spell of delirium and restlessness the patient becomes more and more exhausted; semicomatose and death quickly supervenes.

In patients with hereditary neurotic tendency the uræmic poisoning seems to cause insanity instead of convulsions.

Insanity of cyanosis from bronchitis, cardiac disease, and **asthma** manifests itself with delirium, sleeplessness, false fears, and hallucinations of sight, usually becoming worse at night, and is more common in old persons and those who have a hereditarily weak nervous system.

In **insanity of oxaluria and phosphaturia** hypochondriasis is the main symptom. The mind is depressed, the patient shows a want of energy, and there is great irritability. This form of insanity affects mainly people of the better classes, who have freely indulged in a very rich diet. Under appropriate treatment the symptoms of hypochrondriasis and insanity will disappear. Some writers contend that it is the brain trouble that causes the presence of phosphates and oxalates of lime in the urine.

Insanity of influenza has been noted during certain epidemics of the disease when the poison seems to affect the nervous system most severely. The symptoms during the attack and afterward manifest themselves in great depression of spirits and the loss of volition. Melancholia fre
follows, complicated with lethargy, anæmia, and v
the body. The brain symptoms may arise from

effect of the influenza on the brain cortex or from the weakening effect of the disease on the entire system. The patients show a loss of energy, loss of memory, a loss of interest in life, are of low spirits, crave alcohol, and show symptoms of premature senility. Most cases recover unless the system has been so weakened by the disease that senile degeneration follows.

Rest, tonics, and a change of air are most valuable.

Insanity of Myxœdema.—Most patients suffering from myxœdema are more or less affected mentally. In advanced cases the patient becomes violent, excited, has symptoms of delusions and confusion, and hallucinations of hearing. In milder cases the patient appears mentally enfeebled, suspicious, slow in action and mildly demented. Others show symptoms of mental exaltation with delusions of grandeur followed after years of illness either by partial recovery, or ending in death from general paralysis. Maniacal outbursts are rare. Of late the thyroid treatment has effected some wonderful cures.

Insanity complicating exophthalmic goitre usually appears in the form of acute mania. The maniacal condition may exist with remissions and exacerbations. The patient is subject to fainting fits followed by a gradual loss of power in the limbs. In some cases the symptoms are those of mild mental depression.

If the treatment directed toward the cure or improvement of exophthalmic goitre proves successful, the patient's mental condition shows a proportionate improvement.

The **insanity of lead poisoning** is rather rare. Lead poisoning usually causes some disease of the nervous system. In some instances lead poisoning may cause mental symptoms such as hallucinations, maniacal attacks, and delusions of persecution.

The treatment is directed mainly to removal of the lead from the system; and if successful the mental symptoms are apt to disappear rapidly.

Anæmic insanity is the result of anæmia of the brain. Most cases show symptoms of melancholia, although some

may develop acute mania. The patient is restless, sleepless, becomes melancholic with suicidal tendencies, and finally develops acute mania, which may alternate with depression and fits of weeping.

The majority of patients recover within three months, or as soon as the anæmic condition improves.

Diabetic insanity usually assumes the form of melancholia. Diabetes may cause peripheral neuritis through its toxic effect on the nervous system, and in the same manner may produce insanity through its poisonous effect upon the brain-tissue.

The patient becomes very much depressed, incapable of work, restless at night, has delusions, and as the diabetic symptoms progress so, too, the mental symptoms grow worse.

Treatment directed toward amelioration of the disease may cause temporary improvement in the mental condition of the patient, but an ultimate cure in advanced cases is almost out of the question.

Metastatic insanity may occur in patients suffering from acute rheumatic infection when the rheumatic poison attacks the nervous centres. It may also occur after severe attacks of erysipelas, syphilitic psoriasis, and in patients suffering from chronic ulceration.

This form of insanity usually appears as acute mania, and its curability depends on the curability of the original infection.

Postfebrile insanity is the result of an exhaustion of the vital powers, due to long-standing infectious disease. This form of insanity may follow any of the fevers, such as scarlet fever, smallpox, typhus, typhoid, intermittent. In children idiocy and epilepsy may occur, while in adults dementia of a curable nature may supervene.

Most patients as a rule recover from the fever from which they are suffering. Some weeks afterward mental exhaustion manifests itself. The symptoms are usually subacute and curable.

Postconnubial insanity is usually transitory in nature and caused by mental excitement of marriage and an excess of sexual intercourse.

This form of insanity appears as melancholia with suicidal tendencies, but in some instances it assumes the epileptic form.

Rest, freedom from sexual excitement, and change of air usually effect a complete cure.

Insanity from loss of the special senses may occur as a result of the loss of sight or of hearing.

The patient becomes melancholy, quiet, subject to hallucinations, or delusions.

If the loss of the senses becomes permanent, the mental symptoms in those showing hereditary predisposition to neurotic troubles become generally permanent.

Insanity following surgical operations appears as a sudden melancholia. One meets many cases of acute mania as a result of a removal of a part or of the whole female generative organs. Most cases recover with appropriate treatment.

It seems as if the sudden cessation of the catamenic function would entail a sudden loss of mental faculties, and just as soon as the system becomes accustomed to the loss of the menstrual function just so soon is there a restoration of the normal mental equilibrium.

The **delusions of young children** may occur either as melancholia with violent screaming and all signs of mental depression after severe injuries or operations. In some children after severe fever delusions with hallucinations may follow, especially if the children show a neurotic heredity.

Bromides and cold baths with a bland diet and febrifuges quickly produce a restoration of the normal condition.

Insanity from somnambulism should not be termed a genuine form of insanity, but is closely allied to epilepsy.

Most somnambulists or sleep-walkers have a neurotic heredity.

During the state of somnambulism acts of violence, homicide or suicide, may be committed.

During the normal condition the somnambulist appears perfectly rational, but it is advisable that such patients be kept under strict surveillance as they may either harm themselves or do harm to others.

Phthisical Insanity.—Phthisis is a very common cause of death among insane people, but, on the other hand, the disease itself may produce a form of insanity which is termed phthisical insanity. As phthisis produces a general anæmia, it naturally brings about an anæmia of the brain-tissue, and in those anæmic and with a predisposing hereditary tendency phthisis is apt to produce insanity.

The symptoms develop slowly, and are at first mild in character. Melancholia with mild delusions, monomaniac morbid suspicion, and irritability go hand-in-hand with a loss of weight, loss of appetite, and a gradual development of pulmonary symptoms. The patient becomes sleepless, refuses food, believes himself poisoned, and occasionally has attacks of excitement. He becomes sullen and morose, and is difficult to rouse, and as the local symptoms increase the disease itself takes on a more acute form.

The cure of this form of insanity depends on the curability of the attack of phthisis.

It is surprising to meet with a hereditary tendency to both insanity and phthisis in the same family. Both insanity and phthisis are apt to attack the organism during its period of development.

Hysteric Insanity.—This is a form of insanity that develops in individuals afflicted with hysteria. It appears either in the form of mania or melancholia with morbid sexual symptoms, imaginary bodily ailment, pronounced erotic symptoms, hysteric convulsions, and at times attempts at suicide.

Some patients imagine themselves possessed of some evil spirits, or to be the residing place of the Deity. Others believe that they have given birth to animals. The recovery of these cases depends on the cure of the hysteria, and removal to an asylum effects a cure in two-thirds of the patients.

Insanity of Masturbation.—This form of insanity is the result of an unnatural sexual abuse. While the habit is apt to be practised by most young children, it is more marked and more freely indulged by children of neurotic tendency. In such children this evil habit is persisted in to the degree

of causing a breakdown of body and mind. Most patients suffering from an advanced form of strong masturbational habits are apt to develop insanity as a direct result of free indulgence in this habit, which shows itself in the form of morbid exaggeration, intensely emotional religious ideas, a disinclination to the companionship of others, especially those of the opposite sex, hypochondriacal thoughts, perversion of feeling, followed frequently by attacks of mania. The patient may recover from these attacks, and be cured by strict supervision and active outdoor life, while, on the other hand, dementia with homicidal tendency may develop. Most patients show a general wasting of the system.

Rheumatic and Choreic Insanity.—There appears to be a close relationship between chorea and rheumatism, and this relationship is shown in a marked manner in rheumatic insanity in the fact that the former is a **predisposing cause** of the latter.

The **exciting cause** seems to be an attack of rheumatism, usually of a subacute nature, which has a debilitating effect, especially on those who show hereditary predisposition to mental trouble.

Symptoms.—The patient becomes very absent-minded, and gradually develops marked excitement and choreic movements. Reflex action is diminished, speech becomes incoherent. The heart becomes rapid and at times sluggish on account of the frequency of the choreic movements, and finally becomes impaired. After a while the limbs become paralyzed, the bladder ceases to act, and reflexes are absent. There are hallucinations of sight, touch, and taste, acute delusions with unconsciousness as regards surroundings, succeeded by confused ideas, and high temperature.

Prognosis.—If we can succeed in eliminating the poison from the system, there is a gradual but marked improvement of all the symptoms, and final recovery is simply a question of time.

The **treatment** consists in giving all the latest remedies for rheumatism.

There are some cases of choreic insanity that do not give

the history of a previous attack of rheumatism. These are cases occurring in those suffering from chronic chorea, and in whom the mental status has become weakened from worry over the constantly recurring choreic motions. Such patients should be given drugs that control the chorea, and at the same time should be well fed and undergo antirheumatic treatment.

Insanity due to gout is of rare occurrence, and is chiefly marked by irritability, depression, and a lack of capability for mental exertion.

Melancholia, occurring at the climacteric, may easily change in gouty individuals into symptoms of acute mania. These attacks are either acute, or they become chronic when any treatment directed for the cure of gout seems to be inefficient.

QUESTIONS.

Mention the important rarer varieties of insanity.
What is probably the chief cause of insanity of Bright's disease?
In what type of nephritis does insanity usually occur?
What are the symptoms, course, and prognosis of nephritic insanity?
Discuss the insanity of cyanosis from bronchitis, cardiac disease, and asthma.
What is the mutual relation between oxaluria, phosphaturia, and the nervous system?
What are the clinical features of insanity during oxaluria and phosphaturia?
How may influenza produce insanity?
What periods of influenza may have symptoms of insanity?
Is the prognosis favorable in influenza insanity?
What treatment should a case of insanity due to influenza have?
Is insanity common during myxœdema?
What are the symptoms of insanity of myxœdema?
What special treatment has been of great value in myxœdematous insanity?
What may be said of the clinical features of insanity of exophthalmic goitre?
Discuss fully the symptoms, causes, course, and treatment of insanity of lead poisoning, anæmia, and diabetes.
What is meant by metastatic insanity? Discuss it.
Why does postfebrile insanity occur?
What diseases bear a causative relation to postfebrile insanity?
What are the types of the symptoms, the period of their appearance, and the prognosis of postfebrile insanity?
What can you state as to the occurrence, cause, symptoms, prognosis, and treatment of connubial insanity?
What special form of surgical operations may be followed by insanity?
Give the reasons why surgical operations may be the cause of insanity.
Discuss the delusions of children as the sequels of disease.

What relation to other forms of insanity does somnambulism bear?

What are the hereditary characteristics and the tendencies of the somnambulist when awake and when walking in sleep?

How may phthisis act as a cause of insanity?

What are the symptoms, course, and prognosis of insanity due to phthisis?

What peculiar fact occurs as to the heredity of phthisis and insanity?

What are the causes and manifestations of hysteric insanity?

What determines the cure of hysteric insanity?

What is the importance of masturbation so far as insanity is concerned?

How would you manage a case of masturbational insanity?

What is the relationship of rheumatism and chorea to insanity?

Name the exciting cause of an attack of rheumatic insanity.

What are the symptoms, prognosis, and treatment of rheumatic and choreic insanity?

Discuss gouty insanity.

APPENDIX.

INSOMNIA.

Significance.—Insomnia is not a disease, but a symptom of disease. It may, however, become so active, prominent, and important a symptom as to constitute a condition which merits individual management and treatment. It is for this reason that a separate brief chapter dealing with it has been added.

Definition.—Insomnia is the term employed to denote actual or absolute sleeplessness, and also lack of fully restful sleep, which might be termed relative sleeplessness. The actual sleeplessness may be total in twenty-four hours, or partial in that short periods of restless sleep are obtained. Again, sleep though obtained in the sense that the patient closes his eyes and loses consciousness, is not at all refreshing and does not restore the bodily wear and tear. The patient awakens easily, and usually in the morning declares that he has not slept at all.

Etiology.—The causes of insomnia are usually classified as comprising four types, namely, organic, toxic, primary, and nervous.

The **organic causes** are summed up in such diseases of the brain and nervous system as cause pain, which are exemplified by chronic pachymeningitis, abscess of the brain, tumor of the brain, new-growths of the meninges, locomotor ataxia, and the like.

The **toxic causes** comprise all cases due to poisons circulating in the blood which by irritation—indeterminate as to its precise nature—of the cerebrospinal axis and especially of the cerebrum cause the insomnia. Such diseases are

nephritis, especially chronic nephritis, jaundice, and diseases of the liver which cause jaundice, and acute and chronic infectious diseases, such as typhoid fever and phthisis.

The **primary causes** imply such cases of insomnia as are not secondary to any disease, but are inherent in the brain-condition, namely, depend on insanity. Such insomnias are almost always obstinate in course and pronounced in degree. The forms of insanity having primary insomnia are melancholia (undoubtedly toxic in its essence), mania, paresis, and paranoia (during the stage of excitement in each).

The **nervous** or **simple causes** are present in nervous individuals, and comprise the two conditions of cerebral congestion and cerebral anæmia. Cerebral congestion is typified by the nerve-tire of the student. Overstudy and anxiety bring too much blood to the brain, which produces great activity and a consequent insomnia. Anæmia of the brain acts precisely in the opposite manner. The brain-cells are ill-nourished and hence irritated, and sleeplessness supervenes.

Pathology.—Insomnia being a symptom and not a disease, may be said to have no pathology in the strict sense of the term. The **pathogenesis** of insomnia is very important, because without a proper knowledge of it treatment is either inadequate or faulty.

The **subjective symptoms** depend largely on the cause, and usually add the pathetic complaint of absolute or relative insomnia to the other symptoms of the disease to which the condition is secondary. In primary insomnia, if the patient is still able to detail his other symptoms, he will describe those of melancholia, the milder degrees of maniacal, paretic, or paranoiacal excitement.

In simple congestive insomnia the patient complains of sense of beating in the head, a sense of fulness, occasionally of headache, of muscular irritability, of frequently awakening to find himself uncovered in bed, and the bed-clothes tossed about.

The **objective symptoms** comprise observation of the fact that the patient does not sleep at all, or intermittently and restlessly, as the case may be.

The subject of congestive insomnia may be observed to toss about in bed, to have muscular twitchings, and to be readily awakened. This form of insomnia is usually relative, the person sleeping very irregularly and restlessly.

Course and Prognosis.—Remediability of the cause determines the brevity of the course and the character of the prognosis. Organic cases are rather obstinate and unfavorable. Toxic insomnia disappears with the cessation of the acute primary diseases, but is a grave sign in chronic primary diseases. The advent of insomnia primarily associated with insanity is distinctly significant of severe types of the disease in question. Simple insomnia vanishes when the cause is remedied. Recurrences of simple insomnia are common, because the patients are apt to produce the causative conditions in virtue of their nervous constitutions.

Treatment.—The management of insomnia is very difficult and highly important, because the symptom is so definite and troublesome that many patients are in danger of contracting drug-habits. All the causes of insomnia are practically either obstinate or recurrent, so that the danger of tempting a patient to depend on drugs is either constant or returns with each relapse of his disease. The two principles of treatment of insomnia are therefore: *remove the cause* and *use caution in the employment of drugs.*

In the **organic insomnia** pain is the cause, and should be removed when possible or quieted by various therapeutic measures, with final resort to drugs when other means have failed. Usually treatment of this type of the disease is not very successful.

The **toxic insomnias** require attention to the sleeplessness in addition to the other symptoms. Some of these cases depend upon anæmia, correction of which by tonics checks the insomnia. Thus the danger of hypnotic habits may be avoided. The cause of insomnia in primary cases associated with insanity can not be removed. Powerful sedatives are therefore required, such as chloral hydrate, hyoscine, and morphine.

From the standpoint of likelihood of drug-habit, perhaps

the **treatment of the simple insomnias of cerebral congestion and cerebral anæmia** are the most important because the patients belong to the nervous type who are likely to have recurrences of the underlying conditions, with manifest inclination toward reliance on drugs. The one safe rule in these cases, therefore, is: *Avoid drugs in every instance, try other means first, and resort to drugs only after these have failed.*

A. **Treatment of Congestive Insomnia.**—I. **Nonmedicinal Measures.**—1. Hot or warm general body-baths are very advantageous—stimulate the circulation and restore its balance alike in congestive and anæmic cases. After such hot baths caution must be exercised to get into bed at once and to remain protected against chilling the body-surface in cold rooms or by drafts.

2. Cold spongings, cold shower-baths, cold needle-baths, or cold plunge-baths are available when the hot or warm baths fail to produce the correct result. The reaction to cold water is usually very prompt, and, unless it is depressing, is more favorable than that to warm or hot water. The subject should be rubbed with warm rough towels until the skin is aglow. If he feels rested and quieted, the reaction is proper; if depressed, the treatment is too vigorous.

3. A valuable hydrotherapeutic measure is to have the patient stand for a few minutes ankle-deep in a tub of *hot* water and throw over him a *drip-sheet* from water at 75°–80° F. temperature. This gives the effect of cold reaction. The patient's back and abdomen should be rubbed hard and a general brisk rubdown had immediately after leaving the tub. The effect of this treatment is properly sedative, not exciting or depressing.

4. The cold abdominal pack is valuable. Flannel is wrung out in water at 75°–80° F. and laid in several thicknesses upon the abdomen of the patient. Over the flannel a *dry* towel is placed and itself is covered with oiled silk overlapping widely in order to protect the bed. The whole is then tied or bandaged on rather firmly. The correct effect of this technic is first that of a cold then of a warm poultice.

5. Exercise is a very potent nonmedicinal element in the treatment, and should be carried out in the open air when possible. A brisk walk, horseback ride, or bicycle ride for a half-hour before bedtime, followed by a rub-down, will frequently cure. Exercise with the dumb-bells, Indian clubs, chest-weights, and the like is also very serviceable. Forcible respiration with or without the aid of pulley-weights is an active factor in relief.

II. **Dietetic Measures.**—Frequently a light, easily digestible supper will encourage sleep, when a hearty, rich dinner will cause insomnia by engorgement of the brain-vessels. A few cases of congestive insomnia do well when a light meal is eaten an hour before bedtime, because the process of digestion withdraws blood from the brain and the balance of the circulation is restored so that sleep is possible.

III. The **medicinal agents** available in congestive insomnia are the following. It is well in treating all simple insomnias to employ at least three different drugs in series from day to day, so that the patient can not very well become accustomed to the action of one remedy, then demand it, and finally become addicted to it.

1. Bromides are the best; they should be taken in a rather large dose (grains 20–30) in plenty of water and a half-hour before retiring. Sodium and strontium bromide are first choice.

2. Chloral hydrate is little used, because it is too potent, dangerous, and disorders the digestion. The chloral-habit is easily established.

3. Sulphonal, trional, and other coal-tar derivatives are good hypnotics, but should always be given with a little food, never alone. If they are given alone, they act most potently after the first meal of the next day is taken. Milk or a small luncheon should always be given with these remedies, especially sulphonal. "The expectation of sleep leads to sleep." Hence one may administer rather a large dose of sulphonal, for example, and after an interval follow it with a large dose of sugar of milk, starch powder, or of any other similar *placebo*, to impress the patient that he is "taking

medicines." This same policy is of value when morphine has been given.

4. Other valuable *placebos* are bread pills, variously colored sugar pills, and hypodermatic injections of sterile water or of normal salt solution.

5. Aconite is a valuable circulatory sedative which should be remembered when cerebral congestion is associated with a high-tension pulse.

B. **Treatment of Anæmic Insomnia.**—As a rule in this form of insomnia the patient goes to sleep, but awakens soon afterward, through irritability of the brain by poor nourishment by anæmic blood. The indications for removing the cause are to improve the quality of the blood and to quicken the circulation.

I. **Dietetic Measures.**—A light supper a half-hour before retiring to bed, such as hot milk, broths, milk-punch, and the like, will very frequently promote sleep. The food-stuff thrown into the blood overcomes the anæmia sufficiently for the purpose. The other daily diet should be as nutritious as possible for the restoration of the blood to normal.

II. **Hydrotherapeutic measures** are less directly available than in congestive insomnia, but indirectly aid in adding tone to the system. The same dictum may be uttered as to employing electricity and massage. The most valuable moment for employing hydrotherapeutics in anæmic insomnia is when the patient awakes after the first period of sleep. A cold sponge, bath, or plunge, or a hot bath or other measures, may be of great service.

III. **Medicinal Measures.**—1. Tonics are always indicated for the renewal of the blood, and thus for the indirect cure of the anæmic insomnia.

2. Cardiac stimulants, such as whisky, beer, and strychnine, act directly by increasing the blood-flow to the brain. The temptations to the alcoholic habit require consideration here.

3. Chloral hydrate may dilate the vessels as well as quiet, but should be used cautiously.

4. Amylene hydrate and chloralamide are the most valuable of the recent hypnotics in this form of insomnia.

5. A full dose of strychnine (gr. $\frac{1}{30}$—$\frac{1}{15}$) will frequently so restore the nerve-tone, independently of its circulatory effect, that sleep is possible, whereas without it the overtired condition of the nerves makes sleep impossible. For this purpose it should be given a half-hour before bedtime.

IV. The **plan of hourly awakenings** is often of emphatic and permanent psychic effect upon patients suffering with anæmic or congestive insomnia. It consists in placing a nurse in charge of the patient with directions to observe how much he sleeps, and to awaken him each hour through the night. It requires as a rule only one such night to convince the patient that he does sleep, and usually with this conviction comes the expectation of sleep, which in its turn induces repose. The subject for this treatment should be carefully selected according to his common sense and willingness to enter upon the test in the proper spirit.

C. **Special Cautions as to Certain Drugs in Insomnia.**—1. Opium should be employed only with greatest care and in extreme cases. In **melancholic** and in marked **anæmic** insomnia opium should be administered in divided doses, for example, grain ¼ should be divided into five or ten doses and repeated at short intervals during an hour before bedtime, and stopped as soon as its effect appears.

2. Morphine is given in the same manner—best as the bimeconate—grain $\frac{1}{20}$ repeated three or four times.

3. The tincture of hyoscyamus (℥ss) is sufficient for the ordinary case in which the sedation of the belladonna group is indicated.

4. Hyoscine and hyoscyamine should be reserved for **mania** alone, and are given in divided doses until physiologic action appears. These are powerful, dangerous remedies and judgment is demanded in their use.

5. When gastric exhibition of drugs is not possible, inhalations of ether, cologne, and alcohol in a handkerchief will often suggest and promote sleep.

6. Hypodermatic employment of drugs is efficacious, but seems to stimulate habit-formation more than any other means.

QUESTIONS.

What is the clinical importance of insomnia?

How would you classify the causes of insomnia?

Give several examples of the causes of insomnia under each of the etiologic classes.

What is the pathology of insomnia?

Why is the pathogenesis of insomnia important?

What are the symptoms of insomnia, subjectively and objectively?

What are the course and prognosis of insomnia?

What is meant by primary insomnia?

Describe simple insomnia.

What is the difference between anæmic and congestive insomnia?

What is implied by the term toxic insomnia?

In what circumstances would you expect to have toxic insomnia?

Is insomnia due to toxæmia more important in acute or chronic diseases?

Why is the use of drugs for insomnia to be avoided as far as possible?

What other measures besides drugs may in general be employed in treating insomnia?

Give in full the dietetic, hygenic, and hydrotherapeutic measures of treating anæmic insomnia, congestive insomnia, toxic insomnia, and primary insomnia.

In employing drugs, what hypnotic would you choose for congestive insomnia, for anæmic insomnia, for toxic insomnia, and for primary insomnia?

State as fully as possible the reason for your selection in each case.

What special precautions would you employ in prescribing the following drugs for cases of insomnia: opium, morphine, hyoscyamus, hyoscine, chloral hydrate, hyoscyamine?

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